Clinical Cystoscopy

A Treatise on Cystoscopic Technic, Diagnosis, Procedures, and Treatment

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IN TWO VOLUMES

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TO

MONA

MY WIFE

Charming, unselfish, gracious companion

AND

PHILIP

MY GRANDSON

Whose infant feet and mind
have yet to explore the mysteries
that surround him, and whose life
is just beginning
It is in him I place my hope

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I

ANOMALIES OF NUMBER

(*a*) *Congenital Absence of Both Kidneys*

Congenital absence of both kidneys is a very rare anomaly and is incompatible with life. Many of those referred to in the literature have been in the presence of other gross physical abnormalities. The presence of other gross abnormalities need not necessarily be apparent, but extra-fetal life cannot exist. It is interesting to note that in the majority of instances reported, oligohydramnios was present. This fact tends to substantiate the theory that

the fetal secretion of urine is closely associated with the production of the amniotic fluid. Such fluid would be absent or at a minimum in the presence of complete renal agenesis. Goward reported a case in which complete renal agenesis was found but a normal amount of amniotic fluid was present.

(b) *Congenital Absence of One Kidney*

The presence of a solitary kidney is very important from the clinical angle. The possibility of the inadvertent removal of a congenital solitary kidney is eliminated by our present diagnostic methods. In the presence of a solitary kidney, a complete absence of nephrogenic tissue exists on one side (Fig. 243). Such

Fig. 243—Bilateral retrograde pyelogram showing aplasia of the right kidney and hydronephrosis of left kidney.
(Courtesy of Dr. Elmer Hess.)

a lack of development would result in a complete absence of the kidney, ureter, and the corresponding side of the trigone The suprarenal may be present in its normal site According to Eisendrath, there are four variations of this anomalous development

1 Complete absence of the kidney, the ureter, and corresponding half of the trigone

2 Complete absence of the kidney and ureter and a development of half of the trigone The functioning ureter ends on the opposite side of the trigone

3 The trigone symmetric, two ureteral orifices and a ureter of variable length of the agenetic renal tissue

4 Complete absence of kidney, ureter, and ureteral orifice on one side the opposite kidney ectopic, the ureter ending in the midline of the trigone

Congenital solitary kidney has an incidence of occurrence of relatively 1 1000 The agenesis is more frequently observed in males and the left kidney is more frequently absent than the right Jameson estimates that associated malformations of the genital tract, in either sex occur in relatively 70 per cent of instances The congenital solitary kidney is usually found in the location that corresponds to that of the normal organ The malplaced kidney has been found in the iliac fossa within the true pelvis or along the midline of the body The suprarenal gland is absent on the side of the agenesis in relatively one third of the cases In a case observed by the author, a congenital solitary pelvic kidney was present (Fig 244) Both adrenals were situated in their normal position and possessed a normal blood supply Such a finding tends to prove that the suprarenals are totally independent in their formation and blood supply from the kidneys proper

The congenital solitary kidney is usually somewhat enlarged as a result of a compensatory hypertrophy and it may be lobulated

or otherwise abnormal in form. Congenital absence of one kidney does not incapacitate an individual from the normal physiological functions. The congenital solitary kidney may be subject to disease more frequently than a similar kidney would be under normal circumstances. For this reason, the diagnosis of disease in

Fig 244—Hypogenesis. Lowpower photomicrograph which shows absence of true glomeruli and abortive attempts through hyperplasia by the tubular epithelium to take over glomerular function.
(Courtesy of Dr Lawrence W Smith and Dr Edwin S Gault.)

the presence of a congenital solitary kidney should be accurate and well established before any surgical intervention be undertaken.

Diagnosis: A solitary kidney is subject to the same surgical diseases as the normal kidney. A solitary kidney presents a more serious outlook. Extreme care should be exercised in establishing the diagnosis as well as in the institution of any surgical measures

directed to the solitary kidney The diagnosis of solitary kidney can only be made by a combination of several factors

1 *Complete physical examination*, particularly in the presence of malformation of the genital tract in either the male or female If anomalies of the external genitalia are noted in the female, or malformations of the penis or urethra, or unilateral absence of the testes or half of the prostate or one of the seminal vesicles, is observed in the male, these abnormalities should be considered as important Congenital absence of one kidney should always be borne in mind whenever symptoms referable to the urinary tract are elicited or whenever abnormalities of the genitalia exist

2 *Cystoscopy* The finding of a single ureteral orifice should immediately suggest the presence of a solitary kidney The observation of two normally placed ureters, one of which cannot be catheterized except for a short distance, should immediately suggest the possibility of such an anomaly The appearance on the x ray plate of the radiopaque material as it is halted at varying heights above the ureteral orifice, without apparent reason, should also suggest such a possibility All are important data and are very suggestive of the presence of solitary kidney

3 *Excretory urography* presents important data A normally excreting kidney, either normally placed or in an ectopic position may be noted in contrast to complete lack of any renal excreting tissue on the opposite side The most important evidence is obtained from the individual renal function tests and the individual catheterization of the ureters with subsequent ureteropyelograms Retrograde pyelography should only be considered in those cases without anuria or after anuria has been relieved

Treatment Treatment varies according to the presence or absence of anuria Ureteral catheterization is possibly the most conservative and yet the most advisable measure for the relief of

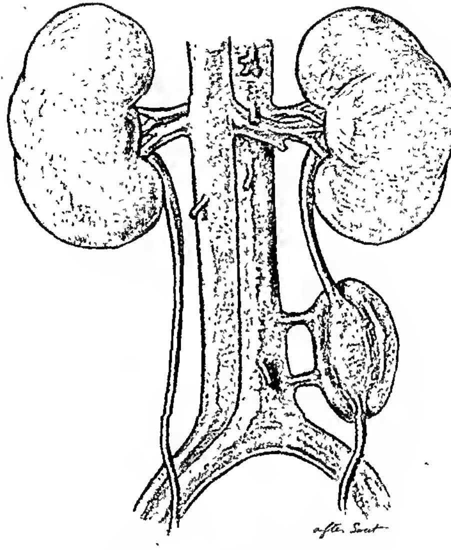

Fig. 245—Supernumerary kidney having a common ureter and ureteral orifice with the normally placed kidney.
(Re drawn from Hinman after Linberg.)

anuria resulting from a temporary obstruction caused by a calculus. In the presence of obstructive anuria, conservative yet heroic measures such as nephrostomy, pyelotomy, or ureterotomy are indicated. The most important single factor in the surgical management of solitary kidney is the establishment of adequate

renal drainage Only conservative measures are indicated as the very existence of the patient rests on the presence and functioning ability of the solitary kidney In the absence of anuria, conservative surgical measures for the relief of nephrolithiasis, with or without an associated hydronephrosis or pyonephrosis, or the relief of the obstruction caused by anomalous vessels should be considered

(c) *Supernumerary Kidney*

Supernumerary kidney is a distinct third kidney with independent capsule and blood supply There is no authentic report of the occurrence of four separate and distinct kidneys The anomaly of supernumerary kidney is rare Only 41 cases have been reported in the literature Such an anomaly must be differentiated from a double kidney or an ectopic kidney with fusion In the presence of a third kidney the ureter may join the ureter of the other kidney on that side (Fig 245) The cystoscopic appearance of the trigone would be that observed when two normal kidneys exist The kidney may have a separate capsule and separate blood supply, a separate ureter and orifice (Fig 246), or the ureter may open extravesically The supernumerary kidney is usually smaller than normal, lying below the normal kidney in the line of the ureter or over the spine It may lie above the normal kidney but such a position is exceedingly rare The presence of a supernumerary kidney may be discovered at autopsy or operation for some unrelated condition as there are no pathognomonic symptoms that would establish its presence As in other renal anomalies, the accessory organ is especially prone to be the seat of calculous formation or infection

Treatment The treatment of the diseased supernumerary kidney does not differ from that applicable to a normally situated kidney diseased in similar fashion

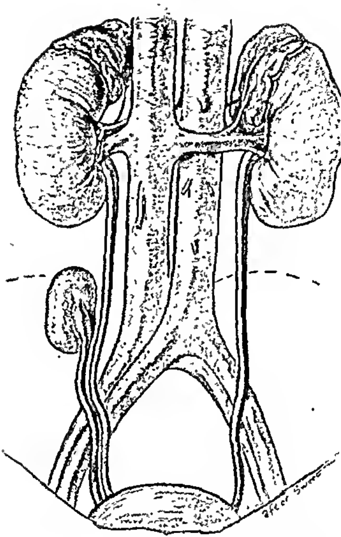

Fig. 246—A distinct third kidney possessing separate ureter and orifice. (Re-drawn from Hinman after Papin.)

Renal Duplication or Double Kidney

Renal duplication or double kidney is a most common renal anomaly. Double kidney is to be differentiated from fused kidney. Double kidney results from ureteral duplication while fused kidney is from congenital fusion of the two kidneys. A double kidney

is usually larger than the average normal kidney and yet it is usually normal in form (FIG 247) The condition may be unilateral or bilateral and is usually associated with duplication of the ureter Duplication of the ureter may be complete or the

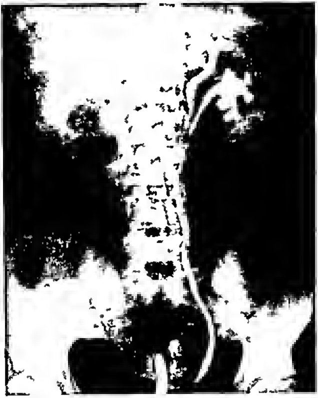

Fig 247—Retrograde pyelogram showing double kidney and double ureter with fusion of the ureter in the upper portion
(Temple University Hospital Acc No 40310)

ureters may unite at any point along their course, to enter the bladder through a single orifice (FIG 248)

The two ureters from a double kidney may run a distinct course each ending by its own ureteral orifice on the trigone of the bladder (FIG 249) Or one ureter may end normally in the bladder and the other ureter may end extravesically in some other portion

of the genitourinary tract. In the male, such an abnormal placement of the ureter may be in the prostatic urethra, in the ejaculatory duct, or in the seminal vesicles. In the female, the ureter may open in the urethra, in the vagina, or in the vestibule of the

Fig. 248—Intravenous urogram showing a ptosis of the right kidney, double left kidney with fusion of the ureter in the upper portion. (Temple University Hospital, Acc No 18904)

vagina. The normal ureter on the duplicated side usually opens in a normal orifice on the trigone of the bladder. Papin found that a high bifurcation of the ureter occurred less frequently than a median bifurcation but that a low bifurcation within three or five centimeters of the bladder occurred most frequently.

The pelves of the kidney are usually placed one over the other although they may be found side by side (Fig 250) The upper pelvis is almost invariably smaller than the lower pelvis although individual function of the two halves may be relatively equal In

Fig 249—Intravenous urogram showing normal left kidney and double right kidney with hypoplasia of the upper half The ureters are separate entering the bladder by two separate orifices
(Temple University Hospital Acc No 39073)

instances of complete duplication of the ureter, the ureter draining the upper segment is usually mesial to the ureter draining the lower half The ureters may be distinctly separate throughout their length or may wind around each other The upper portion of the double kidney is almost invariably the diseased portion

The lower portion is more voluminous and it is more normally shaped. The ureter to the lower portion is usually more normally situated. The upper portion of the double kidney is, from this evidence, the supernumerary or abnormal portion (Fig. 251).

Fig. 250—Intravenous urogram showing bilateral double kidneys. There is immediate fusion of the ureter.
(Temple University Hospital, Acc No. 36832)

It is generally conceded that an abnormal organ is more susceptible to pathological conditions than a normal organ. A very large proportion of double kidneys and ureters will be found diseased. Statistics demonstrate that malformations predispose to disease (Fig. 252). In double kidney and double ureter the

Fig 251—Retrograde pyelogram showing double kidney and complete duplication of the ureters
(*Philadelphia General Hospital*)

presence of hydronephrosis or pyonephrosis is quite common Double kidneys like other renal abnormalities are prone to the presence of pathological changes especially infections calculi hydronephrosis and pyonephrosis Tumors in the presence of half of a double kidney have been repeatedly reported in the literature However infection is the commonest pathological condition observed The next commonest pathological entity is hydronephrosis The presence of calculi is third in occurrence Undoubtedly these three conditions are the result of stasis which is very prone to occur in the presence of ureteral anomalies

Double kidney is usually discovered during study in an effort to establish the cause of the urinary stasis. In many instances the condition produces no symptoms. It is discovered accidentally in the course of routine study.

Diagnosis: The diagnosis of double kidney is usually established by cystoscopy, ureteral catheterization and retrograde ureteropyelography in comparison with excretory urography. For this comparative study it is preferred to begin the study of the

Fig. 252—Retrograde pyelogram showing a double kidney, hypoplasia of the upper half and slight hydronephrosis of the lower half. Two distinct ureters are present.
(Courtesy of Dr. Elmer Hess.)

upper urinary tract through the use of excretory urography Such a study will usually demonstrate or suggest the presence of anomalous development It is a simple matter to bypass the junction of a bifid ureter of a double kidney when catheterizing a single ureteral orifice For this reason a ureteropyelogram should always be made using a catheter possessing a bulbous enlargement sufficient to occlude the ureteral orifice The patient should be placed in a modified Trendelenburg position and the radiopaque medium permitted to flow by gravity along the ureter Both ureters and pelves are demonstrated radiographically by this procedure

Treatment The treatment depends upon those conditions found after complete urologic study The presence of double kidney does not necessitate operative measures

II

ANOMALIES OF SIZE

(a) *Renal Hypoplasia*

Congenital hypoplasia of the kidney is not infrequently seen There are two types

Type I The structure of the kidney resembles in every detail a normally developed organ in miniature The kidney has its own renal pedicle ureter and normally placed ureteral orifice

Type II The structure of the kidney presents only traces of the various components of the parenchyma (glomeruli and tubules) which may only be found by histologic study The renal mass has no specific form or contour The mass has no excretory function The renal artery is usually rudimentary and extends from the aorta to the fibrous thread of tissue The ureter may be present and open in a normal position on the trigone Such a ureter may be patulous for a short distance only and then extend

as a fibrous cord or may be present as a fibrous cord throughout its length.

The type of hypoplasia most frequently observed is that of a dwarfed kidney (FIG. 253). Resembling a normally developed

Fig. 253—Bilateral retrograde pyelogram showing hypoplastic right kidney, normal left kidney.
(Courtesy of Dr. Elmer Hess)

kidney, it is usually found in the kidney bed but is very diminutive in size. Although generally observed to be located in the normal position it may be found to occupy a much lower position and even be adherent in the pelvis to the bladder or surrounding structures. The renal pelvis is usually present but is atypical in form and considerably smaller in size than normal. The ureter,

as previously mentioned, may be partially or completely obliterated or even absent. On section the hypoplastic kidney may present a typical demarcation between the cortex and medulla. These two components are usually observed in a normal ratio proportionately but definitely in miniature as compared with the normal. Histologic sections may reveal the presence of embryonic tubules and glomeruli, while in other fields, sclerotic fibrous tissue and irregularly arranged tubules may be observed. There is a definite tendency to cyst formation.

Clinically, the hypoplastic kidney is subject to the same pathologic conditions as those observed in a normally developed organ and is predisposed to such diseases as hydronephrosis, calculous formation, nontuberculous and tuberculous infection.

Symptoms: The symptom usually portrayed is that of pain. The pain may be colicky in nature but it is generally evidenced as a constant, dull pain in the loin.

Diagnosis: A definite diagnosis of the presence or absence of hypoplastic kidney is imperative. The diagnosis is particularly necessary when surgical measures to the opposite side are contemplated. There are instances recorded in the literature where the opposite, normally functioning kidney has been removed with subsequent death to the patient.

Cystoscopy may or may not reveal important data. The ureteral orifices may be normally situated and the ureters may be patulous throughout. The hypoplastic kidney may have sufficient tubules capable of excreting urine that is normal in every way. In contrast, the ureteral orifice may be noted to be extremely small but this, in itself, is not pathognomonic of the presence of hypoplastic kidney. The catheter may be advanced only for a short distance during ureteral catheterization, the ureter being a fibrous cord above. Under such circumstances, indigo-carmine would not be eliminated from the affected side.

Excretory urography may yield considerable important data as to the presence of a nonfunctioning kidney, or the presence of a rudimentary type of pelvis on the affected side. The most accurate data may only be obtained by retrograde pyelography.

Retrograde pyelography: The catheter is introduced up the ureter in the customary manner. If the ureter is not patulous the radiopaque medium may be returned or regurgitated from the ureteral orifice into the bladder. If the ureter is patulous throughout its entire length, the pyelogram following the injection of the radiopaque solution would reveal the presence of a miniature atypical pelvis in its normal or in an abnormal position.

The importance of an accurate diagnosis of a hypoplastic kidney cannot be underestimated. In the past it has been considered that any kidney capable of concentration of dye on function tests, or capable of excretion of normal urine could, under stress, hypertrophy to such a degree as to sustain life. Such a statement is definitely untrue. This fact is revealed at autopsy in those instances where nephrectomy on the opposite side has been done. Possibly one of the most important aids in making an accurate diagnosis of the presence of a hypoplastic kidney is the use of the intravenous urogram. Because visualization or the complete lack of visualization of a hypoplastic kidney leads to an accurate study of that side. Having proven the presence of a hypoplastic kidney on one side, the opposite side should be subjected to the most meticulous attention before operation is considered.

Treatment: The treatment of an affected hypoplastic kidney does not differ in any manner from that of a similar lesion of a normally developed kidney. However, the treatment of the opposite side may vary considerably. Due to the presence of the hypoplastic kidney, conservative treatment of the opposite functioning kidney, regardless of its condition, must be instituted rather than any radical procedure such as nephrectomy.

Atrophic Kidney

Unfortunately there is no clear distinction between congenital hypoplastic kidney and atrophic kidney Atrophic kidneys whether congenital or acquired are of the utmost importance Individually such a kidney is unable to sustain life Geraghty and Plaggemayer stated that the most pathognomonic sign of atrophic kidney is that the appearance time of excreted dyestuff may be normal but that the total excretion for a given time is much reduced on the affected side Its occurrence is such and its importance is such that as in congenital renal hypoplasia the presence of atrophic kidney should be ruled out if nephrectomy is to be considered on the opposite side

Congenital Renal Hypertrophy

The hypertrophic or compensatory kidney is always noted in those cases of congenital absence or marked atrophy of the opposite kidney The kidney is characterized not only by its increased size but also by its functional capacity as it is compelled to do double duty The incidental finding of an enlarged but otherwise normal kidney should immediately demand examination of the entire urinary tract Removal of this otherwise normal kidney (excepting its size) would invariably terminate fatally when lack of function of the opposite side exists or function is markedly reduced

Symptoms There are no pathognomonic symptoms of renal hypertrophy In the presence of associated pathological lesions the same symptom complex of a kidney of normal size would be noted

Treatment Conservative surgical measures should always be taken Surgical measures depend upon the condition present and the functioning ability of the opposite kidney

III

ANOMALIES OF FORM

(a) *Lobulated Kidney or Fetal Kidney*

Lobulation of the kidney is normal for the fetus and infant but should disappear at the age of four years. The surface of the kidney is segmented by sulci which divide the renal parenchyma in rounded or irregular lobes corresponding to the Malpighian pyramids. Lobulation may persist in adult life and is of no clinical significance.

(b) *Congenital Cystic Kidney*

Congenital cystic kidney will be discussed under the heading of renal cysts.

(c) *Fused Kidney*

Crossed renal ectopia with fusion is a relatively uncommon condition. In a review of the literature, mention was found of only 224 cases. Beer and Hegerman estimated the occurrence of this anomaly to be 1 to 8000 cases. Stewart and Lodge found it only once in 6500 autopsies. Of the cases reported, the right kidney was more frequently displaced than the left; the condition was seen more frequently in males than in females. Usually the displaced kidney lies below the normally placed organ, fusion taking place at the poles of the kidneys (Fig. 254). The ureters remain separate and enter the bladder on their respective sides as the ureter from the displaced organ is usually found to run obliquely across the body to enter the bladder at the normal site. Considerable variations of the fusion between the two kidneys may exist. The kidneys may fuse side to side, end to end, or at a distinct angle (Fig. 255).

Symptoms The presence of crossed renal ectopia does not present pathognomonic symptoms Lithiasis acute and chronic pyelonephritis pyonephrosis and hydronephrosis are prone to occur in anomalies of the kidney

Fig 254—Crossed renal ectopia. Retrograde pyelogram showing the course of the right ureter which enters the bladder at the normal site The course of the ureter can be seen as it traverses the spinal column at an angle

(Temple University Hospital Acc No 3336?)

Diagnosis The diagnosis of fused kidney is made by cystoscopy and retrograde pyelography

Cystoscopy There is nothing pathognomonic in the cystoscopic picture on which a diagnosis may be made

Very little dependence as to a correct diagnosis can be placed

on physical findings or the palpation of the renal mass. The diagnosis is only attained by the use of excretory urography or retrograde pyelography. Of the two methods, retrograde pyelography is more accurate. A more clear outline of the displaced ureter

Fig. 255—Crossed renal ectopia. Bilateral retrograde pyelogram showing hydronephrosis of the upper half of a fused kidney. The left kidney is ectopic. (Temple University Hospital, Acc. No 51294)

and renal pelvis is usually attained more readily than with excretory urography.

Treatment: The treatment of fused kidney varies according to the conditions present in each individual case. Prior to any surgical procedure, particularly heminephrectomy, the function of the other half of the fused kidney must be given careful consideration.

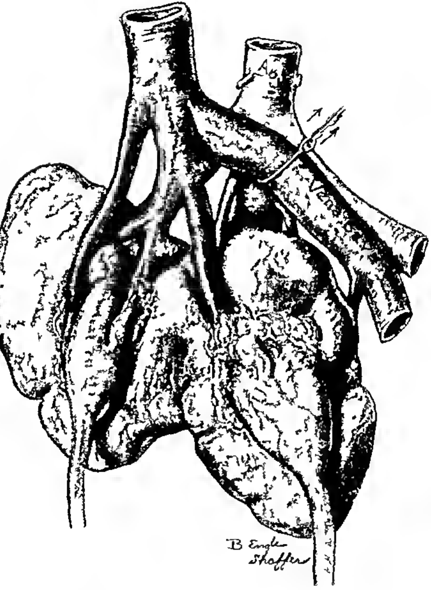

Fig 256—Ectopia of both kidneys with fusion of the lateral surfaces Note the anterior position of the renal pelves and the abnormal arrangement of the renal vessels
(Temple University Hospital Acc No 1568)

1. *Asymmetrical Fusion*

This may take the form of L-shaped kidney, cake kidney, disc kidney, sigmoid kidney, and dumbbell kidney which are formed of unilateral kidney fusion and although they may be found in a midline position (FIG. 256), they cannot be considered a true horseshoe kidney.

The ectopic portion usually occupies the lower portion of the kidney mass. The kidney normally found on the respective side

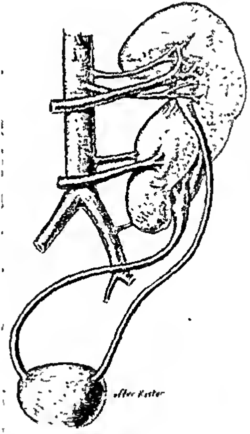

Fig. 257—Sigmoid kidney. The hilum of the lower kidney faces in the opposite direction from that of the upper kidney.
(After Küster.)

is usually in the more elevated position of the mass The variations and malarrangements which may be assumed by the mass are extensive The kidneys may fuse end to end (Fig 257), side to side, at an acute angle with each other, or at right angles (L shaped kidney) (Fig 258) Many variations of the union may exist in the production of different recognized types or forms The ureter on the ectopic side passes diagonally across the spine entering the bladder in a normal position the kidney from which it arises being usually in the ventral position of the renal mass The ureter from the relatively normally situated portion

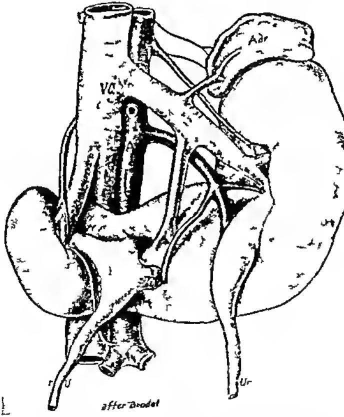

Fig 258—Unilateral horse shoe kidney The left half is larger than the right The ureter of the right kidney is observed coming from the bridge or isthmus (Re-drawn from Brodel after Kuster)

of the kidney mass prescribes a relatively normal course to the bladder, terminating in a normal position on the trigone (Fig. 259). Instances have been recorded in which a definite crossing of the ureters existed. The ureter which would usually enter the bladder on the right crosses to the left, while the ureter usually entering on the left side crosses to enter the bladder on the right. Many variations are possible in such a malformation.

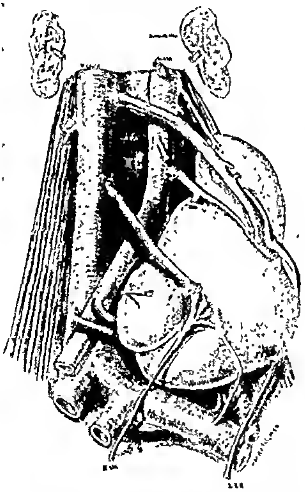

Fig. 259—Unilateral fused kidney. The right kidney is ectopic and is superimposed on the left orthotopic kidney.
(Courtesy of Dr. Harry A. Walmer and *Journal of Urology* 1938, 40 351)

2 *Horseshoe Kidney*

Horseshoe kidney (*ren arcuatus*) is the commonest form of kidney fusion The condition was first described in 1552 by Beranger de Carpie The two kidneys are intimately fused at the similar poles which results in a renal structure that assumes the shape from which its name is derived (Fig 260) It is usual that the two sides are asymmetrical The incidence of occurrence reported by various investigators has varied widely Macalpine,

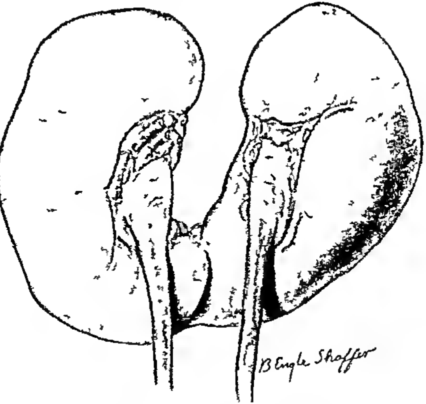

Fig 260—Horseshoe kidney Note median isthmus and anterior position of renal pelvis There is a distinct furrow in the kidney substance caused by the pressure of the ureters on the renal substance

(Temple University Hospital Acc. No 1037)

in totaling the reports of several investigators, found that in a total of 284,656 autopsies, it occurred once in 782 instances. The author, in a survey of 12,029 autopsies at the Philadelphia General Hospital, spanning a period of five years, found that it occurred only 7 times, or 1 in 1718 instances. In relatively 90 per cent of instances of occurrence, the two kidneys are fused at the lower poles, the concavity passing upward. Fusion at the upper poles does occur, although with less frequency, the concavity passing downward. The two kidneys are united by an isthmus which may be a thin, fibrous cord of membranous union or the isthmus may actually be a continuity of the parenchyma of considerable breadth and thickness. The isthmus between the two kidneys usually lies in front of the aorta and vena cava. There are three reported instances in which the isthmus was found to be posterior to the great vessels. The renal mass lies closer to the midline and is approximately two vertebrae lower in position than normally situated kidneys. The isthmus usually lies at the level of the bifurcation of the aorta (FIG. 261). Due to the relative position of the renal mass it assumes a plane that is much more anterior than that assumed by a normally situated kidney.

It is rare that a horseshoe kidney is typically symmetrical. One half is usually larger and more normally shaped than its fellow. Each half of the fused kidney possesses its own blood supply. The vascular supply is erratic and extremely variable. This is of considerable importance because of the danger to which these vessels are exposed at the time of operation. Arterial branches not only arise from the aorta itself but may spring from the common iliac, the internal or external iliac and even from the inferior mesentery artery. The majority of the arteries enter the hilus of the kidney but some may perforate its surface. It has been observed occasionally that a separate artery supplies the isthmus. Such an independent blood supply occurs when the isthmus is composed

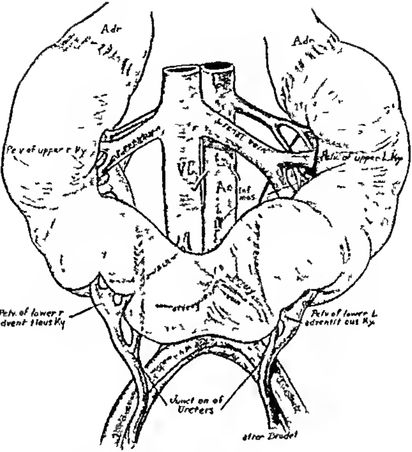

Fig 261—Horseshoe kidney of remarkable type There are four distinct kidneys the upper two lie in an approximately normal situation with each hilum facing median ward those of the two lower kidneys face downward and outward. Each kidney has a separate pelvis and ureter, the ureters on same side unite near the pelvic brim The blood supply of this horseshoe kidney is complex, as shown in the diagram

(Re-drawn from Kelly and Burnam after Brodel.)

of parenchymal substances Papin, in a review of 139 cases of horseshoe kidney, found that the arteries supplying a horseshoe kidney varied in number from one to ten vessels but that three vessels were the most customary number The veins are less irregular in their distribution and usually pass directly to the vena cava, but may empty directly into the iliac veins

Fig. 262—Adenocarcinoma—Horseshoe kidney. Retrograde pyelogram. Note the *malposition of the kidneys. The right half shows abnormal pelvis. Adeno-*carcinoma of right half revealed at operation.
(Courtesy of Dr. Elmer Hess.)

The ureter, in many cases of horseshoe kidney, enters the renal pelvis higher than it does in the normal kidney. The ureters usually lie anteriorly and cross the isthmus in their course to the bladder. Lying across the isthmus, the ureters may cause a distinct furrow in the renal tissue and may show some evidence of being dilated above the isthmus. There is usually no dilatation of the ureters below the isthmus. Hydronephrosis in the presence of a horseshoe kidney is of frequent occurrence (Fig. 262). It undoubtedly results from the presence of the high insertion of the ureter and the pressure exerted by the ureter lying on the surface

of the isthmus and possibly by the occurrence of aberrant vessels (Fig 263)

Symptoms The symptoms produced by horseshoe kidney are varied in scope and intensity Abdominal pain may be due to the

Fig 263—Horseshoe kidney Bilateral retrograde pyelogram The renal pelves lie in a relatively normal position.
(Courtesy of Dr Elmer Hess)

pressure of the isthmus on the great vessels Cardiac hypertrophy may result from pressure of the aorta Edema of the extremities may result from pressure of the isthmus on the vena cava For clinical purposes the symptomatology may be divided into two groups

1. Rovsing's syndrome. This symptom complex was first presented by Rovsing of Copenhagen and attributed to the pressure of the isthmus of the horseshoe kidney upon the abdominal vessels and nerves. The pain is distributed in the upper abdomen and is most marked with change of position from the supine to the sitting position. Occasionally the pain is experienced on assuming the standing or sitting position. For the relief of the pain, bending forward often releases the pressure on the great vessels. An intensification of the pain is experienced upon bending backward.

Horseshoe syndrome. Gutierrez, in an analysis of 25 cases, found abdominal pain a common symptom. He has advanced as the most characteristic point in the clinical symptomatology the horseshoe syndrome which is characterized by three prominent clinical symptoms: (1) Abdominal pain localized in the epigastric or umbilical area; (2) chronic constipation with or without associated gastrointestinal disorders; (3) urinary disturbances manifesting the early clinical signs of chronic nephritis.

2. The symptoms produced by pathological changes existing within the horseshoe kidney are similar to the symptoms that exist in other renal anomalies. There are no pathognomonic signs or symptoms. It is only due to the high degree of efficiency of present urological methods that a diagnosis of horseshoe kidney may be made preoperatively. Even then a diagnosis may be difficult or impossible. The symptoms portrayed by co-existing pathological lesions assume the typical symptomatology found in a kidney that is normal in position and contour (Figs. 264 and 265). The diagnosis of the presence of horseshoe kidney showing definite and typical symptomatology of renal pathology may only be an accidental finding at the time of examination.

Diagnosis: It is occasionally possible in a thin individual to palpate the isthmus of a horseshoe kidney as it crosses the vertebral

Fig. 264—Horseshoe kidney Roentgenogram showing direction taken by the left ureter
(Temple University Hospital Acc No 54534)

column An accentuation of the pulsation of the abdominal aorta may be present These findings may only suggest the presence of horseshoe kidney but it remains for the roentgen ray to establish a final diagnosis A presented history of vague abdominal pain in altering the body position in the presence of a centrally located palpable abdominal mass should be looked upon with suspicion for a horseshoe kidney until proven otherwise

Cystoscopy There is nothing in the appearance of the bladder that is suggestive of horseshoe kidney

Intravenous urography showing the position and contour of

Fig. 265—Horseshoe kidney. Intravenous urogram showing bilateral hydronephrosis of both halves of kidney. Opaque catheters (Fig. 264) reveal the course of the ureters.
(Temple University Hospital, Acc. No. 54554)

the renal pelvis has been of great diagnostic value. The preoperative diagnosis of a horseshoe kidney may readily be overlooked particularly where the kidneys are in a relatively high position and the connecting isthmus is long.

Retrograde pyelography: Bilateral retrograde pyelography affords the greatest diagnostic aid. The pyelographic picture presented usually reveals a renal pelvis that is lower than normal and lying closer to the median line than normally observed. The axis of the two pelves diverges as they pass upward. This divergence

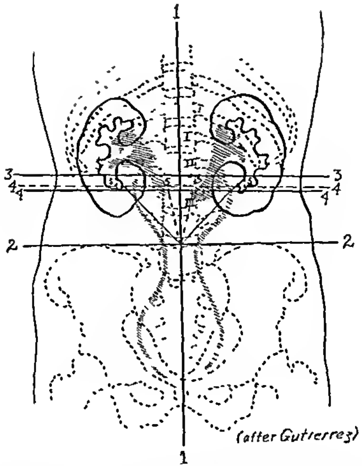

Fig 266—Method of computing the basal angle of a bilateral pyelogram The pelves and kidney shadows in outline represent the normal with a basal angle of about 90 degrees (64 to 103 degrees) The shaded pelves and ureters represent the usual finding in bilateral pyelography of horseshoe kidney with a basal angle (dotted lines) of about 20 degrees (7 to 36 degrees)

Method of computation *1–1 = vertical vertebral line 2–2 = transverse* bisiliac line The point where 1 and 2 cross is the apex of the triangle. 3–3 = transverse vertebral line between second and third lumbar vertebrae 4–4 = horizontal line connecting the two lowest and most internally situated calyces (normal in solid line, horseshoe kidney dotted line) 5 (solid, normal, dotted, horseshoe kidney) projection from 4 to 3 The line between these two points on 3 (5–5, solid, is normal, 5–5, dotted, is in horseshoe kidney) is the base of the triangle

(After Gutierrez.)

led Gutierrez to construct a basal angle on the pyelogram for the diagnosis of horseshoe kidney (FIG. 266). He found that the basal angle was from 7 to 30 degrees in the presence of horseshoe kidney as compared with a basal angle in the normal kidney of 64 to 103 degrees. The presence of a horseshoe kidney may be confirmed by the determination of such a basal angle. It is occasionally possible to outline the kidneys and the isthmus by a plain roentgenogram, but such a finding is usually exceptional. Pyelography will reveal that the pelves are usually everted and tend to radiate away from each other but this is not a universal finding. It has been

Fig. 267—Horseshoe kidney. Retrograde pyelogram. The right half shows *hydronephrosis. The left half exhibits evidence of congenital polycystic kidney.*

(Courtesy of Dr. Elmer Hess)

demonstrated that the pelves may be on the inner surface, the anterior surface or the outer surface of the renal mass (Fig 267) It is only by means of a lateral pyelogram that the plane in which the fused kidneys lie may be ascertained In questionable instances of occurrence the pyelogram should be taken not only from

Fig 268—Horseshoe kidney Bilateral retrograde pyelogram Note the posit on of the renal pelves lying below the brim of the pelvis
(Courtesy of Dr Elmer Hess)

the anteroposterior position but also from a lateral position Lateral pyelography will reveal the displaced pelves lying in front of the spine The ureters prescribe a backward course after emergence from the anteriorly placed kidney, to assume their customary course downward to the bladder Additional information may be gained by retrograde ureteropyelography in ascertaining

the true course of the ureter which may not be well visualized by the excretory urogram (Fig. 268).

Treatment: The horseshoe kidney is frequently subject to complicating pathology as are all anomalous developments. The presence of hydronephrosis, pyonephrosis, calculous formation, infection, acute and chronic, are frequently complicating factors. Abdominal pain as a result of pressure of the isthmus on the great vessels, or the pain resulting from the pull or drag from the misplaced kidney, may necessitate division of the isthmus and nephropexy. The complicating pathology is managed only according to the pathological conditions presented. Once a fused kidney has become diseased, little hope of a permanent cure may be gained except by radical procedures.

(d) *Double Kidney—Duplication of Pelves and Ureters*

The most frequent anomaly of the kidney is duplication of the renal pelves and ureters. The anomaly may be unilateral or bilateral; it may be partial or complete, or may be an association of the two. In duplication, the kidneys possess two distinct pelves and each individually presents a ureter extending to the bladder as a separate channel. In bifurcation, the ureters unite below the kidney to enter the bladder as a single channel (Fig. 269).

In the presence of a unilateral double kidney or complete duplication, two ureteral openings are found on the corresponding side of the trigone. The two ureters are entirely separate throughout but are usually contained within a single sheath.

In the presence of bilateral double kidney, two ureteral openings are found on each side of the trigone.

In unilateral incomplete duplication, the kidney possesses two pelves, the ureters fusing at some point between the kidney and bladder, to open on the trigone by a single ureteral orifice.

Bilateral incomplete duplication is observed less frequently

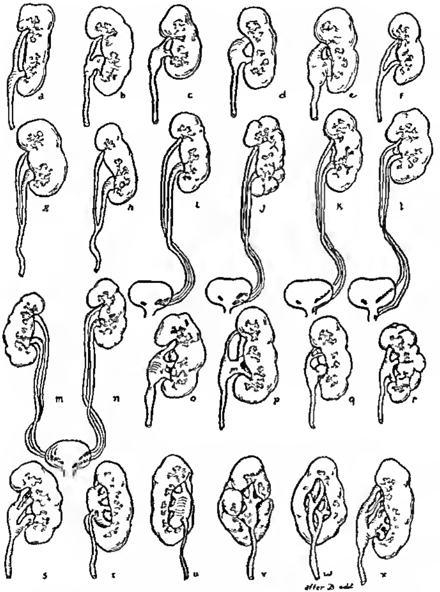

Fig. 269—Schematic drawing showing the possible anomalies of ureters and renal pelves

(Re-drawn from Kelly and Burnam after Brodel)

than the other three anomalous forms, but it does occur. Instances of multiple ureteral orifices have been recorded but the presence of multiplicity of the kidney pelves is lacking. Although the ureters usually lie within a common sheath they may cross and recross each other. It is usual that the ureteral orifice that lies more mesially and caudally, that is, nearer the vesical neck, leads to the upper half of the double kidney. The lower half of the kidney is drained by the ureter that opens in a more lateral and higher plane (FIG. 270). The ureters may lie side by side or

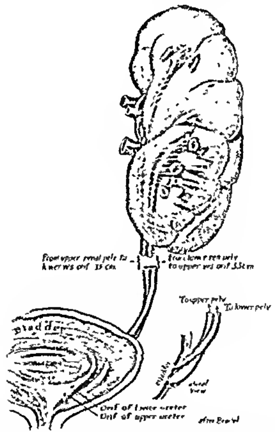

Fig. 270—The most usual type of double pelves and double ureter. The crossing of the ureters is shown in the small diagram to the right. It is usual that the lower ureteral orifice is the outlet of the upper half of a double kidney.
(Redrawn from Kelly and Burnam after Brödel.)

may lie one above the other, or may appear at an angle to each other in their relative positions on the interureteral ridge Duplication of the renal pelves may occur in other anomalies such as horseshoe kidney or fused kidney

Symptoms There are no pathognomonic symptoms of duplication of the kidney The presence of duplication should be suspected in any instance of urinary incontinence or pyuria that persists in spite of all efforts to find the cause in an apparently normally developed upper urinary tract The occurrence of the ectopically ending ureter is frequently seen in this maldevelopment The unsuspected presence of duplication may be demonstrated by a routine x ray or cystoscopic examination

Diagnosis *Cystoscopy* The presence of multiple ureteral orifices is sufficient to make an immediate diagnosis of double kidney An inverted Y formation of the ureter exhibiting two ureteral orifices on one side of the trigone and, after union, continuing as a single ureter to the kidney is an exceedingly rare anomaly

If the ureteral orifices are situated normally on the interureteral ridge the diagnosis of duplication is relatively simple The diagnosis of such a condition may be difficult if fusion of the ureter occurs above the bladder with a single ureteral orifice visible on each side of the trigone

Intravenous urography may reveal the presence of the normally functioning half of the kidney However, as a result of disease or as the result of the nonfunctioning ability of one half of the kidney in the presence of disease, the presence of a double kidney may not be revealed

Retrograde ureterography should be done in every instance of continued pyuria or in the presence of continued symptoms referable to the kidney area The ureteral catheter should be large enough to occlude the ureter and the radiopaque solution should be permitted to flow into the ureter by gravity with the patient

placed in a modified Trendelenburg position. By this means bifurcation of the ureter may be discovered in totally unsuspected instances. A careful search for an ectopically placed ureteral orifice should be made in every instance of continued urinary incontinence which persists in spite of all methods of treatment. A simultaneous ureteropyelogram should be made after catheter-

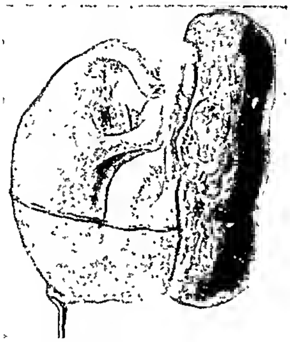

Fig. 271—Extrarenal calices—dorsal aspect.
(Courtesy of Dr L. L. Vereen and *Journal of Urology* 1933, 30 487.)

ization of the ectopically placed ureter and after the catheterization of the normally placed ureter. The ectopically placed orifice frequently communicates with one-half of a double kidney.

Treatment: No treatment is required if the presence of double kidney is accidentally found and no symptoms are evident. The method of treatment of such pathological conditions as nephritis or infection is identical with that of the treatment of similar conditions involving a normally formed kidney. The treatment of the

pathological lesions demanding surgery varies according to the individual conditions present A diseased condition of the kidney may necessitate nephrectomy or heminephrectomy, but in each instance the treatment is governed by the conditions present

(e) *Anomalies of the Pelvis Other Than Duplication*

Anomalies of the renal pelvis may be divided into two groups (1) Congenital hydronephrosis, and (2) extrarenal pelves and calices (Fig 271) As a result of faulty developmental defects there may be a dilatation of the pelvis (which is usually associated with similar dilatation of the ureter) No demonstrable obstructive uropathy may be evident in the entire urinary tract Some investi

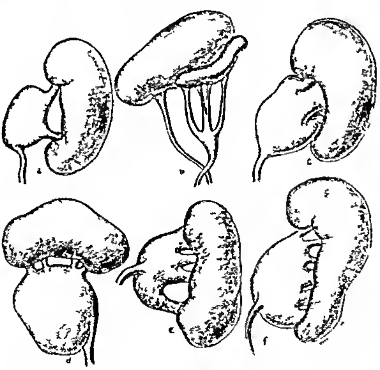

Fig 272—Diagrammatic representation of extrarenal pelvis, (a) after Scholl (b) after Furstner, (c) after von Gaza, (d) after Herz, (e) after Wildbolz, (f) after Eisendrath
(Re-drawn from Hinman after Scheele)

gators believe that such a condition cannot exist without the presence of some obstructive lesion. It has been proven by numerous investigators that hydronephrosis may occur without the presence of an obstructive lesion. The condition is particularly noted in infants and children.

The occurrence of extrarenal pelves and calices is a rather uncommon anomaly (FIG. 272). Such an anomaly may assume considerable importance in the presence of anomalous positions or other gross malformations of the kidney.

Symptoms: There are no pathognomonic symptoms of the presence of either of these anomalies.

Treatment: The treatment depends upon the associated conditions present.

IV

ANOMALIES OF POSITION

(a) *Movable Kidney*

(dropped, floating kidney, nephroptosis)

The normal kidney is not stationarily fixed in position but moves in its bed on respiration or change of body position. The normal range of mobility of the kidney is from two to five centimeters. The right kidney is slightly more mobile than the left. Mobility on one or both sides occasionally becomes greater without apparent cause. The condition is more frequently seen in women than in men. Kelly and Burnham, in a survey of 7853 autopsies, found the occurrence of nephroptosis to be 22.8 per cent in the female and only 2.1 per cent in the male. Mobility, in itself, is not a pathological entity. Mobility only becomes pathological when (as a result of the position of the kidney) pathological changes are induced by such mobility and when there is

a disturbance of normal function Obviously these disturbances of function are more frequently associated with extreme mobility

Two clinical groups of movable kidney must be differentiated (1) Mobility without disturbance of function, (2) mobility with disturbance of function As the mobility becomes extreme, permitting the kidney to come to rest in a horizontal position (providing the pedicle is sufficiently long) the ureter becomes redundant and exhibits evidence of torsion and kinking As a result, the ureter may show varying degrees of dilatation This dilatation is also manifest in the renal pelvis

Symptoms The condition may be symptomless and may only be discovered during routine examination The kidney may be palpated at various levels or may accidentally be demonstrated by roentgen ray (FIG 273) There may be severe pain on the side of the ptosis located in either the iliac fossa or the upper abdominal quadrant or both Such pain is known as Dietl s crisis, a syndrome first described by Dietl of Vienna The pain may be associated with reflex symptoms such as nausea vomiting, abdominal distention, and rapid pulse The pain usually radiates downward along the course of the ureter During the attack there may be a marked decrease in the urinary output but following subsidence of the pain a large amount of urine is usually voided Occasionally hematuria is present during the attack or immediately following The crisis may be attributed to a temporary hydronephrosis and with its disappearance the pain and symptoms subside In the more chronic or permanent forms of nephroptosis, the acute nature of the attacks is not apparent but presents more of the picture of a chronic hydronephrosis with or without infection The same is true here as in any form of stasis of the urinary tract, stasis predisposes to infection For that reason, in every instance of persistent pyuria a careful examination for nephroptosis should be made

Diagnosis: *Cystoscopy:* There is nothing cystoscopically pathognomonic of renal ptosis. Although the history is important, little pertinent data may be truly gained by cystoscopy. In instances of Dietl's crisis, a similar history may be obtained in instances of

Fig. 273—Ptosis of kidney. Retrograde pyelogram showing a mild hydronephrosis in the presence of a ptosis of the right kidney.
(Temple University Hospital, Acc. No. 46326)

biliary calculus, intestinal obstruction, or acute appendicitis. The evidence of a mass which may be palpated when the patient is lying down or standing up, although important, cannot truly demonstrate the intrinsic state of the kidney.

By far the most important data to be obtained are by urographic

study, by both intravenous and retrograde methods Not only can the kidney function be ascertained but the position of the kidney may be established by the roentgenological studies To demonstrate the presence of nephroptosis it is imperative that the roentgenograms be taken in the horizontal and also in the upright positions Compression should not be exerted on the abdomen while either the intravenous or the retrograde urogram is being made It may be with some difficulty that the entire ureter is demonstrated by routine procedure If a stiff ureteral catheter is used any tortuosities or kinks of the ureter may be eliminated A ureteropyelogram done with the patient in the erect position may demonstrate the kinks The ureter should be catheterized with a bulbous catheter which occludes the ureteral orifice The entire ureter and kidney pelvis may be more readily visualized during distention by the radiopaque solution

Treatment: It is generally accepted that any instances of symptomless nephroptosis should not be subjected to operative procedures In those instances where there is ureteral obstruction due to kinks, angulation, the production of a hydronephrosis and a continuance of infection which resists all treatment, nephropexy is indicated To subject every patient to nephropexy who possesses a movable kidney is subjecting many of them to needless surgery

(b) *Renal Ectopia*

Simple unilateral renal ectopia is a condition in which one or both kidneys are arrested in their normal upward migration and may become congenitally displaced and fixed in some abnormal position (Fig. 271) One kidney is located in a normal position, the other kidney being in a position that may vary from a high lumbar level to a position within the bony pelvis (Fig 275)

It is possible for both kidneys to be displaced Both kidneys

Fig. 274—Anatomical specimen showing marked anomalous arteries of a pelvic kidney.
(Temple University Hospital, Acc. No. 3750.)

may be on their respective sides of the body but in a position considerably lower than is normal.

It is also possible that one kidney may migrate to the opposite side of the body, coming to rest below the normally situated kidney, there being no fusion between the two kidneys, or it is possible that the kidneys may be fused. Such a fusion may be from end to end, side to side, assuming the typical characteristics of a disc, shield, or L-shaped kidney.

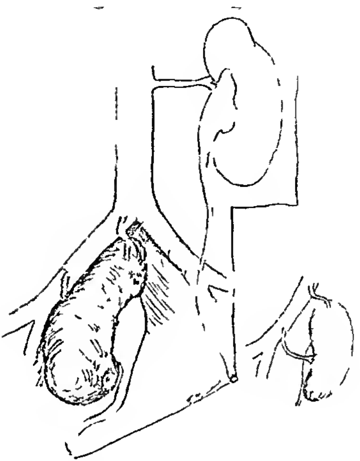

Fig 275—Pelvic kidney right Normal left kidney Note one anomalous artery entering parenchyma another passing posterior to the kidney to enter the hilus normally
(Courtesy of Dr Elmer Hess)

Ectopic kidneys occur relatively one in 1000 cases occurring equally in both sexes There is normally a change of the blood supply as the kidneys ascend in their normal development If, for any reason the ascent of the kidneys is arrested, the arterial supply becomes permanent at the time that the arrest occurs (Fig 276) For this reason the blood supply to an ectopic kidney is anomalous and may arise from the aorta, the iliac, or the

mesentery arteries. A simple ectopic kidney may be found in a higher position than normal, or at a position midway between its natural position and the iliac crest. More frequently the kid ney may be found lying at the crest of the ilium or in the iliac fossa, but most frequently within the true pelvis (FIG. 277). Due to the position of the kidney the ureter may be short and tortuous but usually opens in a normal position on the trigone. An anomalous position of the ureteral orifice is possible in association with an anomalous position of the kidney.

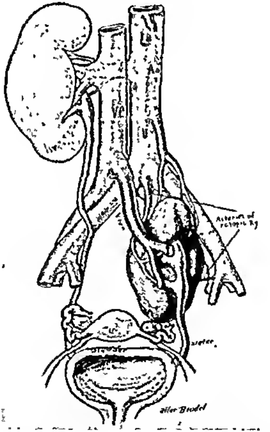

Fig. 276—Ectopic kidney on left side, with a normal right kidney. Note anomalous arterial supply.
(Re-drawn from Kelly and Burnam after Brödel.)

Symptoms: A kidney misplaced in position is not necessarily diseased. Every known pathological condition found in a normally situated kidney may be found in a congenitally ectopic kidney. There may be no symptoms referable to such an ectopically

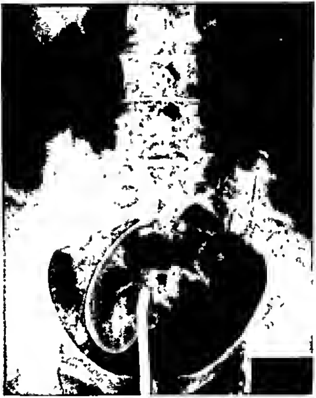

Fig. 277—Pelvic kidney. Retrograde pyelogram showing abnormal position of right kidney. Note short ureter and blunting of the calices.
(Temple University Hospital, Acc. No 38593)

placed kidney but as the drainage is usually faulty, infection, calculus, and hydronephrosis are common. Due to their aberrant position injury is more likely, particularly in the female during pregnancy as a result of pressure from the gravid uterus. As a result of its abnormal position, vague, obscure, abdominal symp-

toms may be present or, as a result of the position of the kidney, symptoms may be referred to the generative organs.

Diagnosis: The diagnosis of ectopic kidney, regardless of its position, may be readily established by excretory urography, retrograde pyelography, or a combination of the two methods.

Treatment: If the ureter is tortuous, considerable relief may be gained by ureteral dilatation and renal pelvic lavage. These procedures may be supplemented by urinary antiseptics. If surgery is necessary the same surgical procedures may be used as though the kidney was normally situated. The one important factor is the recognition of an ectopic kidney before operation is undertaken. Surgical procedures in the presence of renal ectopia are usually more difficult than upon the normally placed kidney, due not only to the relative position of the abnormally placed kidney but also to its abnormal blood supply and the presence of extensive adhesions between the kidney and the surrounding structures. Usually the kidney is not surrounded by perirenal fat when the ectopic organ lies within the bony pelvis. Any adhesions that may occur would be directly between the surrounding organs and the ectopic kidney.

Although crossed renal ectopia without fusion exists it is commoner to find such an anomaly in which fusion is present. The kidney, during its upward migration, crosses to the opposite side of the body. The ectopic kidney usually comes to rest at a position relatively adjacent but beneath the normally placed kidney. The ureter crosses the spine diagonally to enter the bladder in a normal position. Due to the close proximity of the two kidneys it is almost impossible to ascertain whether fusion does or does not exist. As the result of the abnormal position of the kidney the blood supply is usually anomalous. The ectopic kidney is more frequently subjected to infection, lithiasis, hydronephrosis, pyonephrosis, and pyelonephritis than is the normally situated kidney.

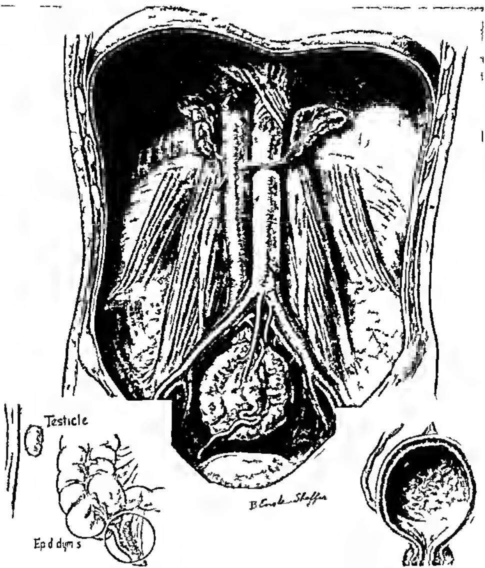

Fig 278—Artist's drawing showing position and blood supply of solitary pelvic kidney Adrenals are normally placed Insert on left shows position of testicle on abdominal wall and epididymis lying in mesentery of appendix. Insert on right shows appearance of bladder with one ureteral orifice only

(L E McCrea and Jour al of Urology 1942 48 58)

Solitary Pelvic Kidney

Congenital solitary pelvic kidney (congenital solitary pelvic renal ectopia) is rare (Fig. 278). The anomalous kidney appears as a solitary intrapelvic organ lying in the midline of the body near the promontory of the sacrum. The condition was first described by Hanow in 1830, and to date only 36 cases have been reported in the literature. Stevens, in an exhaustive review of the literature, estimated that congenital solitary pelvic kidney occurred once in 22,000 autopsies. Because of the anatomical position of the solitary pelvic kidney, pathology referable to the urinary tract reaches a greater proportion in earlier life than with the usual kidney anomaly. Only one case of those reported was older than 47 years. The case was reported by Houtappel; the patient attained the age of 69, dying as a result of pneumonia following pyelotomy for the removal of a calculus at the ureteropelvic junction.

Developmental defects of the genitalia were present in relatively 50 per cent of all the cases reported, a percentage which is a great deal higher than with the more usual kidney anomalies. The blood supply of the intrapelvic kidney is abnormal and may be derived from the iliac arteries or from the aorta.

In a case seen by the author there were two vessels furnishing the blood supply of the kidney (Fig. 279). The larger was five millimeters in diameter arising at the bifurcation of the aorta, dividing into three branches before entering the kidney substance. A right and a left branch entered at the upper poles of the respective segments of the renal parenchyma. A third branch reached the hilum entering the renal parenchyma to the right of the pelvis. A second main arterial supply was a vessel three millimeters in diameter arising from the left common iliac artery.

Fig 279—Photograph of sol tary pelv c k dney as removed at autopsy
L E M C ea and Jou nal of Urology 194 48 58)

This artery was short measuring three centimeters in length and entered the upper pole of the left component of the renal mass. The patient, a man 33 years of age, died of uremia. Microscopic examination of a section of the kidney removed at autopsy revealed the presence of immature glomeruli in some areas and a marked degree of damage to the mature glomeruli (FIG. 280).

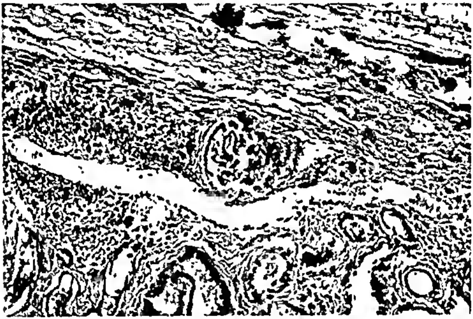

Fig. 280—Microphotograph showing an immature glomerulus in the cortical region.
(L. E. McCrea and *Journal of Urology*, 1942, 48 58)

Some of the glomeruli presented dense bands of adhesions between the tuft and the capsule. Others were completely obliterated by fibrous balls having a laminated appearance. There was also tubular degeneration with atrophy and dilatation, thus revealing that although the individual was 33 years of age, a condition of hypogenesis was present (FIG. 281). The case was of further interest as the only genital anomaly portrayed was an un-

Fig 281—Microphotograph showing inflammatory involvement of glomerulus with pericapsular thickening Interstitial fibrosis and tubular dilatation are also seen.

(L E McCrea and *Journal of Urology*, 1942, 48 58)

descended testicle on the right side. At autopsy it was found that the testicle was adherent to the anterior abdominal wall and the epididymis was found lying in the mesentery of the appendix. Both adrenals were present and were normally placed and showed normal development, having a normal blood supply.

Symptoms: The symptoms of congenital pelvic kidney may be those of a chronic nephritis. These symptoms are usually associated with an alteration of the blood pressure and the excretion of large quantities of urine of low specific gravity. Pain, when present, appears low down in the pelvis and in the median line Hematuria may or may not be present

Diagnosis: *Cystoscopy* reveals the presence of a single ureteral orifice opening in a normal position on the ureteral ridge. The lat-

ter is observed to fade away gradually, to become lost in the bladder mucosa. A second ureteral orifice cannot be seen. The catheter can only be introduced for a short distance up the straight and unhindered ureter. The diagnosis can only be made by retrograde pyelography (Fig. 282).

Treatment: Due to the fact that there is only one kidney and that kidney lies in an abnormal position the treatment should always be of the most conservative nature.

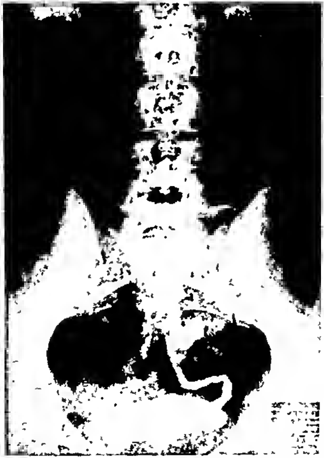

Fig. 282—Solitary pelvic kidney. Retrograde pyelogram showing left kidney lying within the true pelvis. Note short, tortuous ureter.
(Courtesy of Dr. Elmer Belt.)

(c) *Abnormal Rotation of the Kidney*

Abnormal rotation of an otherwise normally developed kidney may be frequently observed (Fig 283) Anomalous rotation is common in fused ectopic or malformed kidneys The rotation of the kidney may be incomplete as during the course of the upward normal migration the kidney normally rotates around its longitudinal axis If this rotation is incomplete or fails to occur, the pelvis and hilus of the kidney may be found in an anterior position Occasionally excessive rotation occurs The hilus is found

Fig 283—Retrograde pyelogram showing a dilated, twisted ureter The kidney is rotated and is hypoplastic
(Temple University Hospital Acc. No. 63063)

to lie posteriorly, the pelvis and ureter descending behind the kidney or along its external margin. Anomalous rotation, whether incomplete or excessive, may be responsible for obstructive changes in the kidney (FIG. 284).

Diagnosis: The most important clinical feature of this condition is the preoperative recognition of the anomaly. Anomalous blood vessels or adhesions may hold the kidney in its relative position. The recognition of this anomalous position may best be established by ureteropyelography. Such a roentgenogram may reveal the calices directed toward the midline of the body in a ventral direction or in both directions. The importance of such

Fig. 284—Intravenous urogram showing normal right kidney: the left kidney is rotated.
(Temple University Hospital, Acc. No 30426.)

Fig 285—Retrograde pyelogram showing rotation of the right kidney
(Philadelphia General Hospital)

findings should never be overlooked in the interpretation of such pyelograms Abnormal rotation may exist alone or in the presence of other anomalies, particularly where fusion of the kidneys has occurred (Fig 285)

V

Anomalies of Renal Blood Vessels

Arterial Side: Anomalous arteries supplying the normally placed kidney are commonly observed These anomalous arteries are of

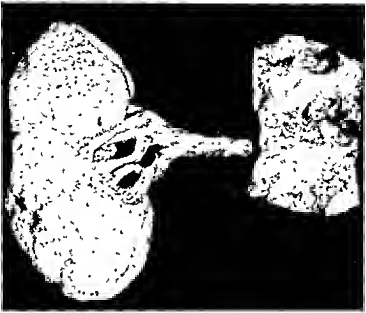

Fig. 286—Abnormal arrangement of renal artery. Six separate branches are present.
(Temple University Hospital, Acc. No 3302)

great variety and distribution (Fig. 286). They are of importance to the surgeon in that by their presence they increase the hazards of renal surgery as well as the frequency with which they produce directly, or indirectly, a hydronephrosis. It is a generally conceded dictum, in the normally formed and normally placed kidney, that single renal arteries without polar branches are normal and that accessory vessels are abnormal regardless of their origin (Fig. 287). Aberrant or anomalous vessels, either in origin or termination, are the rule in fused, ectopic or otherwise malformed or malpositioned kidneys.

Eisendrath, in a study of 418 cases, found that anomalous arteries from the aorta entered the upper pole of the kidney 18 times, the aberrant artery entering the upper pole from the main renal artery in 68 instances. He also found that the anomalous vessel entered the lower pole from the aorta in 38 instances and only

one instance in which the anomalous vessel arose from the renal artery to enter the lower pole

Although these anomalous arteries arise more frequently directly from the aorta or the renal artery, they may arise from other

Fig 287—Retrograde pyelogram showing a hydronephrosis of left kidney Note sharp angulation of ureter caused by an aberrant artery
(Temple University Hospital Acc No 42278)

arteries such as the common and internal iliac, the spermatic, the medial sacral hepatic and inferior mesenteric arteries Two or more renal arteries may enter the hilus from the aorta or from other vessels or both right and left renal arteries may arise from a single aortic trunk

A wide variation in the origin of the blood vessels supplying the kidney may exist. Usually the arteries entering the upper pole of the kidneys are of clinical interest at the time of operation. Those arteries entering the lower pole and those entering

Fig. 288—Retrograde pyelogram showing a hydronephrosis of right kidney. Below the renal pelvis can be observed a distinct shadow transversely across the ureter. At operation an aberrant vessel was found.
(Temple University Hospital, Acc. No. 52631)

the hilus abnormally may be responsible directly or indirectly for the production of a hydronephrosis as the ureter may be angulated or kinked (FIG. 288). The hydronephrosis produced presents the same clinical phenomena as the hydronephrosis produced by any other cause. The hydronephrosis so produced is

usually observed in younger individuals than a similar condition produced by other causes (Fig 289)

The Venous Side Under normal anatomical conditions there is one renal vein lying in an anterior position Anomalous veins

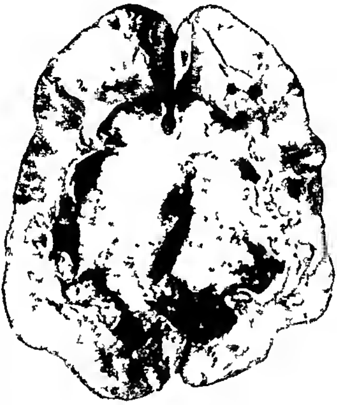

Fig 289—Kidney Hydronephrosis caused by an aberrant artery (*Temple University Hospital Acc No 4173*)

occur less frequently than anomalous arteries Although the anomalous arrangement of the veins may vary considerably the most frequent abnormality is the renal vein lying posterior to the renal artery The main renal vein may bifurcate after leaving the vena cava one branch passing in front and a second branch

passing behind the pelvis. An anomalous vein may be found running from the upper pole or from the lower pole of the kidney. A large vein and a larger artery from the vena cava and aorta respectively may pass behind the pelvis, making the pelvis assume

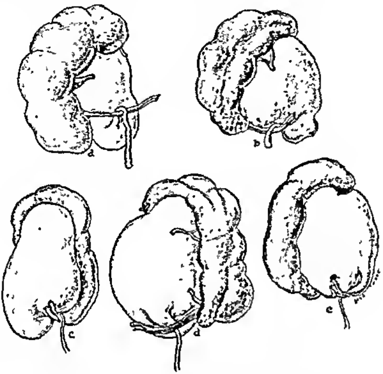

Fig. 290—Schematic drawing showing how an accessory blood vessel can produce obstruction.
(After Andler Re-drawn from Hinman after Sweet.)

an anterior position instead of the usual posterior position, the kidney otherwise normal in position and contour.

Diagnosis: Symptoms are not necessarily present in every instance of anomalous formation of the blood vessels. Many instances are observed either at operation or at autopsy. The occurrence of anomalous vessels may never have been suspected. The most important data may be gained by ureteropyelography in those instances producing symptoms (Fig. 290). A typical distention of

the kidney pelvis and a filling defect at the ureteropelvic junction are usually demonstrated. Similar information may be gained from excretory urography providing the damage to the kidney is not so excessive as to interfere with the excretory ability of the kidney.

Treatment: The treatment of anomalous blood vessels is one affording relief of obstruction whenever it occurs. Preferably, surgical relief of the obstruction should be done before destruction of the kidney occurs.

XIX

RENAL FUNCTION TESTS

Although tests of renal function do not usually constitute a part of cystoscopic procedure, the two subjects are so closely allied that a description of those tests most frequently employed is included. A considerable proportion of urologic surgery is in one way or another dependent upon the functional ability of the kidneys. Total renal function is seldom if ever employed in the immediate association of cystoscopy. Individual renal function always requires cystoscopy.

Considerable important information is to be gained by renal function tests. The answers to such pertinent questions as the total functioning ability of both kidneys or the individual function of each kidney are obtained. To subject the patient to a kidney operation without complete knowledge of the functioning ability of the opposite side is folly. In this day of modern equipment and accuracy of diagnosis, such carelessness is inexcusable.

It is not always the answer desired to determine the functioning ability of the diseased kidney, but what is of greater importance is the accurate estimation of the functioning ability of the supposedly good kidney. It is for the answers to these two questions that renal tests are employed before surgical measures are instituted. Total renal function tests are those applied to the function of both kidneys. Differential, individual, or separate renal function is the estimation of function of each kidney. The ureteral specimens are collected separately. Cystoscopy and cystoscopic procedures are necessary to collect these individual specimens.

The tests of kidney function are divided into three groups: (1) Urinary tests; (2) blood tests; (3) combined tests of blood and urine.

As may be readily seen, many of these tests hold no reference to cystoscopy or cystoscopic procedures but are important in urologic practice. The first group of tests is made following the administration of a definite and measured amount of foreign substance capable of being excreted by the kidney. The amount of excretion of this substance in the urine is measured by chemical or visual methods over a given period of time. Any foreign substance used in excretion tests must be managed by the kidney in a manner similar to that which the kidney employs in the elimination of the waste products of the body.

The tests should represent the work of the kidney in elimination of these foreign substances as a comparative excretory capacity in its ordinary work and should provide a true criterion of its function.

THE PHENOLSULFONPHTHALEIN TEST

The phenolsulfonphthalein test for total renal function was first introduced by Rountree and Geraghty in 1910. Phenolsulfonphthalein is a bright red crystalline powder, soluble in water, and is nontoxic Ever since the introduction of phenolsulfonphthalein, it is possibly the most widely used test for the estimation of renal function activity. The tests depend upon the administration of the foreign yet inert substance, phenolsulfonphthalein, and the measurement of its quantitative excretion in the urine.

The test is a relatively simple one. (1) The patient empties the bladder and is given 240 cubic centimeters (8 ounces) of water in order to procure active diuresis; (2) 10 to 20 minutes after the ingestion of the water, one cubic centimeter of phenolsulfon-

TESTS OF KIDNEY FUNCTION

1. Urine
    - Excretion
        - Total renal function
          Phenolsulfonphthalein
        - Individual renal function
          Phenolsulfonphthalein
        - Chromocystoscopy
          Intravenous urography
            - Phenolsulfonphthalein
            - Indigo-carmine
    - Concentration
        - Specific gravity tests
          Mosenthal, Fishberg
        - Urea concentration
2. Blood–Retention
    - Nonprotein nitrogen
    - Urea nitrogen
    - Creatinine
3. Combined
   Blood and Urine–Clearance
    - Urea
    - Creatinine

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   - Nonprotein nitrogen
   - Urea nitrogen
   - Creatinine
3. Combined
   Blood and Urine–Clearance
   - Urea
   - Creatinine

phthalein solution containing six milligrams of the dye, is injected intravenously or intramuscularly, (3) if the intravenous route is used, the bladder is emptied at the end of one hour following the injection, either by voiding or by catheter, (4) the bladder is again emptied at the end of the second hour. If the intramuscular route is employed ten minutes are permitted for absorption of the dye. The bladder is emptied at one hour and ten minutes and at two hours and ten minutes, following the injection of the dye. (5) the urine specimens are kept in separate containers. (6) a ten per cent solution of sodium hydroxide is added to each of the specimens until a maximum red color is produced, (7) each of the specimens is diluted to 1000 cubic centimeters with water. (8) a portion of the diluted specimen is compared with a standard Dunning colorimeter. The Dunning colorimeter consists of a series of ampules containing dilutions of phenolsulfonphthalein from 5 to 100 per cent of the standard solution that is 6 milligrams in 1000 cubic centimeters equals 100 per cent. The total quantity of urine excreted by the kidneys during this test does not materially interfere with the accuracy of the test. The urine specimen must be further diluted before estimations of excreting function are made. When kidney function is greatly *impaired, the output of phenolsulfonphthalein* varies more or less directly with the volume of the urine excreted. A normal individual eliminates 40 to 60 per cent of the quantity of the dye injected during the first hour and 20 to 25 per cent in the second hour. A total of 60 to 85 per cent of the dye should be excreted normally during the two hour period. Any percentage of elimination totaling more than 55 per cent in the two hour period is considered to be within normal limits by many investigators.

The technic of the test has been recently revised. The time of the test has been shortened to 15 and 20 minute estimations of ex

cretion instead of the hourly intervals. The minimal elimination to be considered as normal, following intravenous administration of the dye, is 28 per cent in the first 15 minutes.

Poor or inadequate renal function, when associated with high nitrogenous retention in the blood, is indicative of marked parenchymal damage. Restoration of renal function, as manifested by higher phenolsulfonphthalein excretion, is necessary before surgical procedures can be safely undertaken.

The one great disadvantage of the phenolsulfonphthalein test is that it is rendered inaccurate in the presence of hematuria. Waters suggests the following for the colorimetric estimation of phenolsulfonphthalein in the presence of hematuria. To the specimen is added 10 to 15 cubic centimeters of concentrated sodium hydroxide. The mixture is heated. Alkaline oxyhemoglobin is formed, hematin is split off, a dark brown solution remains. When the mixture is warm, ten cubic centimeters of magnesium chloride solution is added, then agitating rapidly, the mixture is heated almost to boiling. It is then diluted to 1000 cubic centimeters, filtered, and compared with the standard colorimeter for reading.

INDIVIDUAL RENAL FUNCTION TESTS

The estimation of individual function of the two kidneys necessitates the collection of the total output of each kidney for a given period of time.

Both ureters are catheterized following introduction of the cystoscope in the usual manner. A whistle-tip ureteral catheter is passed to the level of each kidney pelvis. The flow of urine from each catheter is observed. If each catheter does not emit the characteristic drip, it is adjusted or irrigated so that it will function properly. The cystoscope is removed, permitting the catheters to

remain *in situ* The catheters are again observed to ascertain if the urinary flow is maintained If the flow from one catheter ceases, the urine is probably passing around the catheter into the bladder It is often possible to reestablish the flow by adjustment of the catheter or by injecting sterile water through the lumen of the catheter to dislodge any obstruction that might be in the eye of the catheter Exactly one cubic centimeter of phenolsulfonphthalein solution (0 6 milligrams) is injected intravenously The appearance time of the dye is noted as it drips from the catheter The appearance time is estimated by collecting the urine from the catheter as it drips directly into a test tube containing a saturated solution of sodium bicarbonate The characteristic Bordeaux red color results The normal appearance time following intravenous injection is from one to five minutes

Following the appearance of the dye, specimens are collected from each kidney for a period of 15 minutes The specimens should be collected in two test tubes labeled right" and ' left' respectively The percentage of excretion is estimated by the standard Dunning colorimeter The normal kidney excretes 15 to 25 per cent during the first 15 minutes and from 5 to 15 per cent during the second 15 minute period

CHROMOURETEROSCOPY—CHROMOCYSTOSCOPY

Chromoureteroscopy is the visual estimation of the appearance time of dyestuffs from the ureteral orifices after intravenous or intramuscular injection For such procedure, phenolsulfonphthalein or *indigo-carmine is generally used*

Methylene blue has been used but its use has not been attended with great success Following intramuscular injection the appearance time of the dye in the urine is normally 30 minutes It is sometimes excreted as a colorless derivative The use of methy

lene blue has been discontinued because of the long time necessary for appearance and the occasional colorless excretion.

Phenolsulfonphthalein is used by many cystoscopists not only for the percentage estimation of function, but also for chromocystoscopy. Sodium bicarbonate solution or boric acid solution is used as the bladder distending medium instead of sterile water. When the alkali, sodium bicarbonate, is used, a typical Bordeaux red color is observed as the phthalein is ejected from the ureteral orifice. If boric acid solution is used, a deep orange color will be seen. The normal appearance time of phenolsulfonphthalein is one to five minutes.

Indigo-carmine was introduced by Voelcker and Joseph in 1904 as a differential renal function test. Its use has been supplanted to a marked degree by phenolsulfonphthalein. For some unexplained reason, indigo-carmine elimination fails in some pathological conditions, notably tuberculosis, even in spite of variable and relatively large amounts of remaining renal tissue.

Occasionally, its intravenous administration is followed by a rather severe reaction. This reaction is characterized by pallor, rapid feeble pulse, varying degrees of shock, pain in the chest, and dyspnea. Although these reactions are sudden and alarming, the patient responds well to the administration of adrenalin chloride (0.6 to 1 cc.—10 to 15 minims) and external heat.

Indigo-carmine is administered by intravenous injection of five cubic centimeters of an 0.4 per cent solution (prepared ampule).

Indigo-carmine is used principally for the visible appreciation of renal function. Attempts have been made to use the dye routinely for percentage estimation of renal function. Such attempts have not been too successful because of serious technical difficulties. An important use is for the localization of a ureteral orifice in a greatly distorted bladder, or malposition of the ureteral orifice. The principal use of indigo-carmine, as it is used today, is

for the visible estimation of the appearance time of excretion. Normally, the appearance time is from three to five minutes. Much may be learned in observing the manner and intensity with which the dye is ejected from the ureteral orifice. Under normal conditions, the dye is ejected from the ureteral orifice as an intense blue stream with considerable force. As the force of the stream of ejection is lost, the blue color may be seen to diffuse rapidly in the distending medium. On the other hand, a forceful ejection of a stream of faint concentration reveals a poorly functioning kidney without ureteral obstruction. In contrast, if concentration is good but the stream seeps slowly out of the ureteral orifice, partial ureteral obstruction is suggested.

Beginners in cystoscopy will often become confused by a swirl of blue in the distending fluid. The blue dyestuff does not appear to come from the ureteral orifice under observation, but from the opposite normally functioning kidney. The blue may be of such concentration as to obscure vision completely in a relatively short time. This difficulty may be overcome by catheterization for a short distance of the ureter of the normally functioning kidney. By so doing, the deeply colored urine is carried through the catheter outside of the bladder. Continued uninterrupted observation of the ureteral orifice on the affected side may be accomplished. If catheterization of the ureter is desired, the intense blue dye may be flushed away by opening the irrigating valve of the cystoscopic sheath, permitting the fluid to wash away the dye as it is ejected. It is deemed unwise to administer the indigo-carmine before introduction of the cystoscope in those instances where exploration of the bladder is to be done. Examine the vesical cavity then have an assistant administer the dyestuff.

Intravenous urography, as a means of determining renal function, is a test of valuable aid and is discussed at length under that heading.

MOSENTHAL TEST

Repeated determination of the urinary specific gravity as a test of kidney function was originally suggested by Hedlinger and Schloyer in 1914 and later modified by Mosenthal.

The test is simple but the rules of application must be rigidly enforced if the test is to be of any value. The one important and only restriction is that no food or fluid of any type is permitted between meals or during the night. As much food or fluid is permitted at each mealtime as is desired by the patient.

A time schedule of the test is outlined for the patient in the following manner. Immediately before breakfast, at 8:00 A.M. the bladder is completely emptied and the urine discarded. The average breakfast is eaten by the patient. The urine is collected in separate, properly labeled containers, at two-hour intervals during the 12-hour day from 8:00 A.M. to 8:00 P.M. The night urine from 8:00 P.M. to 8:00 A.M. is collected as a single specimen.

The normal response is one in which the specific gravity varies ten points or more from the highest to the lowest point of concentration. The specific gravity of the night urine should be 1018 or more. The total amount of urine excreted during the day period of 12 hours should be three to four times as great as that eliminated during the night period.

As the renal lesion progresses, the signs of diminished function are:

1. The ability of the kidney diminishes and it is unable to concentrate solid substances.
2. The amount of urine voided at night increases.
3. The specific gravity of the urine becomes fixed at lower levels than is normal.

FISHBERG CONCENTRATION TEST

This test is similar to the Mosenthal test Both tests depend upon the determination of the specific gravity of the urine The day before the test the patient is permitted to eat a usual luncheon No fluid is taken after lunch At the evening meal meat potatoes vegetables pie or gelatin is permitted No fluid is permitted during this meal nor is any more food or fluid permitted until the test is completed Before retiring the bladder is emptied and the urine discarded The first voided urine is collected on awakening in the morning The specimen is kept in a separate bottle The patient remains in bed for an hour longer and then collects a second specimen which is also placed in a separate bottle A third specimen is collected after another hour and placed in another separate bottle The specific gravity of each of the three specimens is accurately determined The highest reading is taken as the patient s maximum It is important that the urine be at the temperature for which the urinometer was designed and calibrated A correction of 0 001 must be added to the specific gravity for each degree the temperature of the specimen is above the calibration temperature likewise a subtraction of 0 001 must be made for each degree below the calibration temperature

The specific gravity will exceed 1022 if the kidney function is normal and unimpaired In severe renal impairment the reading will be 1010 or less In mild degrees of impairment the reading will be between 1010 and 1022

UREA CONCENTRATION TEST

The urea concentration test (MacLean) was originally considered as an estimation of renal function in nephritis However its use has been included in those tests supplementary to surgical

preparation in determining the functional ability of the kidneys.

Technic of Test: All fluids are withheld for eight or ten hours prior to the beginning of the test. The bladder of the patient is emptied. After emptying the bladder, 15 grams of urea in 100 cubic centimeters of water are administered by mouth. After one hour, the bladder is emptied and the urine discarded. The second and third hourly specimens should contain two per cent or more of urea as determined by the hypobromite method of determination.

The test is based upon the fact that urea in normal concentration is from 2 to 2.5 per cent. Normal kidneys may concentrate urea to four per cent or more, if necessary. The test may also be applied to differential kidney tests. The ureters are catheterized during the second or third hour and individual specimens are collected after the administration of urea. A sufficient quantity of urine is collected from each ureter for the estimation of the percentage of urea.

BLOOD—NONPROTEIN NITROGEN

Tests of retention are based on the accumulation in the blood of certain products whose presence is indicative of renal decompensation. Of these retained waste products, nitrogen retention is the most important index of renal function. Nonprotein nitrogen retention occurs only when chronic disease of the kidney is well advanced. It is therefore important, as a prognostic value, to ascertain such retention before operative measures are instituted. Urea, uric acid, ammonia, and creatinine are the nonprotein nitrogenous bodies and contain all the blood nitrogen after removal of the protein content. Marked retention of nonprotein nitrogen is of grave significance in the presence of definite renal impairment.

Two substances urea nitrogen and creatinine are the most important present in the total nonprotein nitrogen retention in the blood Urea nitrogen is largely of exogenous origin It is therefore an important index of the excreting ability of the kidney to rid the body of waste products of protein metabolism The fasting normal value of urea nitrogen varies from 9 to 17 milligrams per 100 cubic centimeters of blood Urea nitrogen determination was one of the first blood tests employed in urology and the one which is possibly more universally used than any other at the present time Upon this test rests the prognostic value of prostatectomy in many instances Retention of 20 milligrams or more of blood urea nitrogen must be taken into consideration and carefully weighed before surgery is contemplated If the urea nitrogen is below 20 milligrams per 100 cubic centimeters of blood the patient is generally regarded as capable of undergoing prostatectomy successfully If the urea nitrogen is found to be as high as 30 milligrams it is a positive indication for proper bladder drainage to reduce the nitrogen retention before surgical procedures are introduced It is true that a high nitrogen retention resulting from obstructive uropathy does not have the grave significance of a nitrogen retention of relative value resulting from true nephritis Furthermore in the retention resulting from obstructive uropathy something material can be done to lessen the retention more than when the retention results from nephritis There is frequently a marked disproportion between the elimination of phenolsulfonphthalein and the degrees of nitrogen retention It has been definitely established that phenolsulfonphthalein will be excreted in a relatively high percentage of elimination for a long period of time after the blood shows nitrogen retention Of the two tests the estimation of nitrogen retention is by far the more accurate of kidney functional ability

CREATININE

Creatinine is normally present in the blood in a concentration of from 1 to 1.5 milligrams per 100 cubic centimeters. Being of endogenous origin, it is not subject to marked fluctuation. It is therefore a more accurate indicator of renal function or dysfunction than either urea nitrogen or total nonprotein nitrogen. A rise of creatinine value of 3.5 or 4 milligrams per 100 cubic centimeters is indicative of definite renal impairment. This is true because the kidney eliminates creatinine more readily than urea. Creatinine values will remain within normal limits long after the urea values show considerable rise.

BLOOD UREA CLEARANCE TEST
(Van Slyke)

The blood urea clearance test has proven itself to be one of the most accurate tests for determining kidney function. The principles of the test are those measuring the excreting ability of the kidney as compared with the blood urea concentration.

The rate of elimination of urea in the urine is directly proportional to the blood urea content if the urine volume eliminated exceeds a certain limit—two cubic centimeters a minute. The standard clearance has been estimated at 54 cubic centimeters of blood freed of urea per minute in adult life.

The routine procedure for the collection of the specimens is as follows:

The patient is instructed not to engage in vigorous exercise prior to the test. The preceding meal, preferably breakfast, should be moderate in amount. Coffee should be prohibited because of the possible stimulating effects of caffeine. The most desired time to do the test is between breakfast and lunch. It is at this time

that excretion is least liable to fluctuate One of the chief errors of the test lies in the possibility of partially emptying the bladder The hourly specimens must be the total of the urine excreted The bladder must be emptied either by voiding or by catheterization At 10 00 A M the bladder is emptied and the urine discarded The patient may be given a glass of water At 11 00 A M the bladder is emptied the urine is collected and labeled specimen No 1 At this time a five to ten cubic centimeter sample of blood is collected from a suitable vein The patient is given a second glass of water A second specimen of urine is collected one hour later and so labeled One of two formulae is used in calculating urea clearance from these data If the volume of urine excreted is greater than two cubic centimeters a minute the maximum clearance is calculated If the volume is less than two cubic centimeters per minute the standard clearance is calculated The concentration of urea in the urine and blood specimens is calculated in milligrams per 100 cubic centimeters The urea clearance and percentage of total renal function are then estimated by the following formulae

C = Maximum clearance (75 cc)	C_s = Standard clearance (54 cc.)
B = Blood urea concentration	U = Urea concentration in the urine
V = Urine volume	

1 If the urine volume exceeds two cubic centimeters per minute the maximum clearance is calculated by the formula

$$C_m = \frac{UV}{B}$$

To obtain the percentage of normal function multiply C_m by the factor 1 26

2 If the urine volume is less than two cubic centimeters per minute the standard clearance is calculated by the formula

$$C_s = \frac{U\sqrt{V}}{B}$$

To obtain the percentage of normal clearance, multiply C_s by the factor 1.76.

Interpretation: Patients showing clearances of 75 per cent of the normal have no impairment of renal function. Clearance values ranging between 75 per cent and 50 per cent of the normal are considered in the doubtful range. It has been demonstrated that a reading of 52 to 56 per cent may occur before phenolsulfonphthalein elimination is diminished. Results below 50 per cent are indicative of renal impairment. Values below 20 per cent are indicative of renal failure. Phenolsulfonphthalein elimination will show a marked diminution. Values of five to ten per cent are usually indicative of approaching uremia.

XX

INJURIES TO THE KIDNEY

Injuries to the kidney are very infrequently observed This is a surprising fact when it is considered that the organ is so vascular and friable Undoubtedly the protection afforded by its retroperitoneal position and normal mobility does much to guard this organ from trauma

Renal injuries may be classified into two groups

1 Subcapsular or closed
2 Open wound

Either of these types of renal injury may result from direct trauma, indirect trauma, muscular action, gunshot or stab wounds In civil practice injuries resulting from direct violence are the most frequently observed (Fig 291) Direct violence to the kidney undoubtedly results from the frequency with which automobile or industrial accidents occur Custer, in an experimental study, was able to produce a rupture of the kidney by direct violence He concluded that the kidney acts like a ball of fluid, the impact being transmitted in all directions simultaneously

Spontaneous rupture of a normal kidney is not known Trivial trauma to the kidney, in the presence of some pathological lesion, is often sufficient to cause a rupture The right kidney is more frequently affected than the left This fact is undoubtedly due to the position of the right kidney which is normally slightly lower than the left.

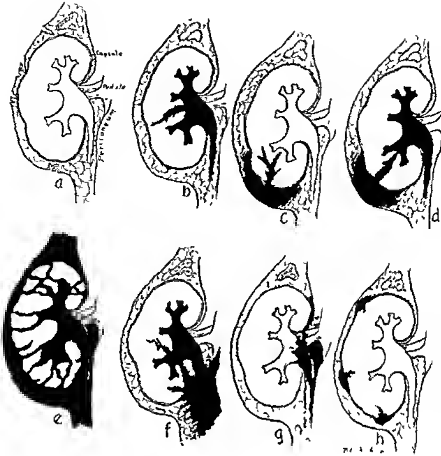


Fig. 291.

A. Rupture of fatty capsule, contusion of kidney.
B. Rupture of parenchyma of kidney, bleeding into pelvis.
C. Rupture of parenchyma of kidney without communication with excretory apparatus.
D. Rupture of parenchyma of kidney and capsule with communication with pelvis.
E. Multiple rupture with fragmentation.
F. Rupture of kidney parenchyma and renal pelvis with extravasation intraperitoneally.
G. Rupture of vascular pedicle and tearing off of ureter.
H. Slight multiple ruptures of parenchyma with subcapsular bleeding, but without communication with the excretory apparatus.

1 Subcapsular or Closed Injuries Subcapsular or closed injuries to the kidney may vary considerably and may be classified as

(a) Contusions
(b) Lacerations or rupture
(c) Dislocations or rotations

(a) *Contusions* Contusions may involve only the perirenal fatty capsule with the formation of a localized hematoma, or the

Fig 292—Rupture of the kidney Note fissure extending into parenchyma and the presence of extensive blood clot.
(Temple University Hospital Acc No 1113)

kidney itself may be traumatized with localized areas of ecchymosis.

(*b*) *Lacerations or rupture:* These may be of trivial nature. The rupture may be extensive and may involve the parenchyma and the ureter and renal pelvis, or the parenchyma and pelves. If the parenchyma alone is lacerated, a subcapsular hematoma usually results, the hematoma varying proportionately to the extent of hemorrhage. Laceration of the parenchyma and renal pelves results in the formation of a hematoma plus urinary extravasation in the subcapsular space (FIG. 292).

(*c*) *Dislocations or rotations:* Dislocation of the kidney is possible rather than probable as a result of violence; a clinical point that would be rather difficult to prove. Nephroptosis may be apparent in many individuals. It would be very difficult to say whether this mobility was due as a result of trauma or the kidney may have been a ptotic organ before injury occurred. Rotation of the kidney is possible although improbable. If trauma is of sufficient violence to cause the kidney to rotate, it would seem probable that the violence would be great enough for the kidney to be torn from its pedicle.

2. Open Wound: Rupture or laceration may result either from violence of great intensity or in instances of marked degeneration of the kidney. If the parenchyma and the renal capsule are ruptured or lacerated, a perirenal hematoma and urinary extravasation occur. The rupture may well be to the extent of peritoneal laceration and extravasation within the abdominal cavity. The laceration may assume many degrees and variations. The kidney may be roughly bisected in either its horizontal or longitudinal plane, which may extend into the kidney pelvis. The lacerations may assume a stellate appearance, so that urinary extravasation and hemorrhage into the surrounding tissues always occur.

Gunshot or stab wounds, although infrequent in civil life, fre-

quently occur in wartime These wounds are generally associated with other grave injuries As a rule, the other abdominal viscera will be simultaneously injured, especially the stomach duodenum, colon, liver or spleen Such extensive injury usually necessitates immediate operative intervention, not only for the renal injury but likewise for the injury to the abdominal viscera

Rupture of a previously diseased kidney is of considerable importance from the industrial standpoint All efforts should be exerted to establish the presence of any type of underlying pathology before attributing the rupture wholly to the result of an accident It may be extremely difficult to establish that a previously diseased kidney existed before accident

Spontaneous rupture of a previously diseased kidney does occur Although rare, cognizance of such a possibility should be borne in mind Spontaneous rupture of a normal kidney does not occur As a result of the weakened diseased walls in hydronephrosis, any increase of tension may be sufficient to produce a tear of the remaining parenchyma with urinary extravasation into the surrounding tissues

Symptoms· There are no pathognomonic symptoms of renal injury although renal injury may present very definite classical symptoms *A renal injury is frequently associated with injury to* the neighboring viscera or with associated fractures of the bony structure In such instances the renal injury may be completely overlooked The symptoms of renal injury, existing by itself, are those of shock, muscle rigidity on the affected side, pain and hematuria A mass in the loin, increasing in size, is occasionally observed Urinary dysfunction is not uncommon

Shock varies with the intensity and extent of injury and the amount of blood lost

Muscle rigidity on the affected side, involving not only the muscles of the back and loin but also abdominal rigidity, may be

noted. The rigidity may be so extreme as to prevent accurate palpation.

Pain is always present. It may be localized at the costovertebral angle; may be generalized over the abdomen; may radiate across the back to the shoulder or downward to the hip or sacrum.

Hematuria is nearly always present. It may vary in amount but is usually constant. Gross hematuria following injury may not necessarily indicate a renal laceration, nor does the absence of hematuria preclude serious renal injury. It is impossible to estimate the amount of renal damage in relation to the amount of blood passed. If there is no hematuria present, such an absence may be caused by a severance of the ureter; a blood clot occluding the ureter or the blood escaping into the peritoneal cavity or into the perirenal tissues. However, one fact is almost proof conclusive. If the patient seems but slightly shocked immediately following a renal injury, later relapsing into profound shock, it is safe to assume that profuse bleeding has occurred.

A mass in the loin may be apparent immediately following injury, or a mass may be observed to appear gradually and increase in size. In such instances, the mass may be limited by Garota's fascia within the perirenal tissues and comprise both blood and extravasated urine. In the presence of an increasing mass resulting from hemorrhage and from extravasation of urine, the temperature will become septic in type within the course of a few days. Such a temperature results from the necrosis of tissue in the presence of the extravasation of urine.

Urinary dysfunction may occur as a complication of renal injury. Such dysfunction may result from altered excretion, obstruction to the excretion of urine, or a combination of the two. Following injury to one kidney, its fellow may develop a reflex anuria. Such an anuria may be transitory and may exist from a few hours to several days depending to a degree on the extent of

shock Persistent reflex anuria may continue to a point where it is fatal Complete persistent anuria may result from bilateral renal rupture or the rupture of a normal kidney whose fellow has undergone some pathological destruction or to the injury of a single kidney whether it be congenital or acquired

In the presence of obstruction to excretion the urine may be prevented from being normally voided although the injured kidney may continue its function normally or be somewhat impaired Complete severance of the ureter would permit an extravasation of urine into the surrounding tissues ureteral obstruction by a blood clot or angulation would produce a hydronephrosis Urinary retention may result from extensive intravesical clotting so that normal voiding is impossible

Diagnosis *History* The history of trauma with associated hematuria pain and muscular rigidity of the affected side although not conclusive is an important point of diagnosis Repeated palpation for the localization of an increasing mass in the loin may also give valuable information Repeated estimations of the hemoglobin and red blood cell counts are important in determining a continued blood loss

Cystoscopy This is not contraindicated except in those instances where the injury is so extensive that immediate operative measures are indicated By cystoscopy it is possible to establish the source of bleeding By the use of intravenous administration of indigo-carmine it is possible to estimate the excreting ability of the affected kidney In instances of suspected ruptured kidney ureteral catheterization is definitely contraindicated

Excretory urography Possibly the most definite and greatest amount of informative data procurable is furnished by excretory urography (Fig 293) The functioning ability of the unaffected kidney may be estimated The urographic shadow produced by the extravasation of the opaque medium into the subcapsular tis

sues and renal parenchyma may be shown when the laceration extends into the calices or renal pelvis. Retrograde pyelography is definitely contraindicated because of the possibility of increasing the hemorrhage as well as introducing infection into the area of traumatized tissues.

Fig. 293—Rupture of left kidney. Intravenous urogram showing abnormal collection of radiopaque medium outside renal pelvis.
(Temple University Hospital, Acc No 41227)

Prognosis: The prognosis of renal injury depends on the extent of injury. Uncomplicated minor injuries of the kidney, which comprise the majority, usually recover under expectant treatment without operation. In the more serious injuries, the prognosis depends to a degree on how promptly surgery is instituted. The type of surgical procedure is dependent entirely upon the condi-

tion present in each individual instance whether it be a nephrectomy or surgical repair. Renal injuries associated with extensive trauma to the abdominal viscera are usually fatal.

Treatment: The first immediate measure is to combat the shock. This is best accomplished by a blood transfusion or the infusion of normal saline. If there is failure of a definite reaction to such procedure and the shock becomes more profound, open operation and exploration are indicated. The great majority of renal injuries may be treated by supportive measures and watchful waiting.

INJURIES TO THE RENAL PELVIS

Trauma or injury to the renal pelvis is uncommon. Injury or rupture may occur as the result of crushing wounds in the presence of similar trauma to the kidney. The pelvis may rupture spontaneously. Pelvic rupture is frequently the result of instrumental trauma by ureteral catheters, bougies or dilators, or the result of injudicious filling of the renal pelvis during retrograde pyelography

Symptoms: The symptoms portrayed are not unlike those of rupture to the kidney proper. The extravasation of urine occurs into the perirenal tissues and if great force has been applied, rupture into the peritoneal cavity is possible. The rupture usually takes place on the posterior wall of the pelvis, due to the anatomical arrangement.

Diagnosis: The diagnosis is best made by intravenous urography. Such procedure will disclose the extravasation of the opaque media into the surrounding tissues. It is unwise to do a retrograde pyelogram for fear of introducing infection.

Treatment: Treatment of injury or trauma to the renal pelvis is similar to that of injury to the kidney proper.

XXI

HYDRONEPHROSIS

Hydronephrosis is a term that implies retention of urine accompanied by dilatation of the renal pelvis and its calices with subsequent atrophy of the parenchyma (PLATE LXIV). The condition is usually the result of obstruction. The normal capacity of the renal pelvis is from 4 to 8 cubic centimeters. Seldom does the normal capacity ever amount to 12 or 15 cubic centimeters. A greater retention than 15 cubic centimeters may be interpreted as a dilatation of the renal pelvis. Hydronephrosis may be classified as small, medium or large according to the amount of fluid retained. Hydronephrosis, beginning primarily with dilatation within the renal pelvis and calices, results in parenchymal atrophy in ratio to the extent of back pressure. A hydronephrosis may be aseptic or may be infected. When infection occurs a hydronephrosis differs from a pyonephrosis. Pyonephrosis is a definite necrosis of the parenchyma as a result of ulceration and cellular destruction, rather than by pressure atrophy. A hydronephrotic kidney may possibly return to function if the obstructive lesion is eliminated before the parenchyma is entirely atrophied. This is in absolute contrast to pyonephrosis. In pyonephrosis there is no possible chance of a return to function following destruction of the parenchyma. An infected hydronephrosis may undergo secondary atrophy in the presence of a severe pyelonephritis, to become pyonephrosis.

The primary or fundamental cause of hydronephrosis is obstruction. The obstruction may be:

1 *Mechanical* Resulting from intrinsic or extrinsic pressure obstructing the normal outflow of urine Such an obstruction may occur at any point along the entire urinary tract from the ureteropelvic junction to the urethral meatus

2 *Dynamic* Resulting from neurogenic or muscular dysfunction of some part of the urinary tract

The mechanical causes of obstructive uropathy are numerous and varied The causes may be classified as congenital or acquired but regardless of their nature or position secondary changes in the kidney invariably occur

MECHANICAL CAUSES OF URINARY OBSTRUCTION

Preputial

- Phimosis
- Paraphimosis
- Tumors in preputial cavity
- Stones in the preputial cavity

Penile

- Tumors of the penis
- Inflammatory masses in the penis
- Periurethral abscess

Urethral

- Congenital or inflammatory stenosis of the meatus
- Atresia
- Stricture (congenital or acquired)
- Diverticulum (congenital or acquired)
- Congenital valves of the posterior urethra
- Congenital enlargement of the verumontanum
- Calculus tumor cysts of the posterior urethra
- Urethrocele
- Fistulae

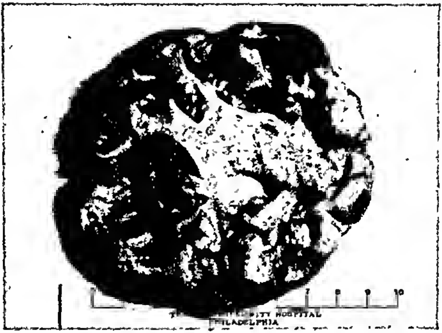


PLATE LXIV—Hydronephrosis. Color photograph showing the thin parenchymal layer and dilated pelvis resulting from an occluding calculus.

Bladder and Bladder Neck:

Congenital anomalies of the bladder
Contracture of the vesical neck (congenital or acquired)
Hypertrophy of the vesical neck
Spasm of the vesical sphincter
Hypertrophy of the interureteral ridge
Vesical diverticulum
Tumor, benign or malignant
Calculus

Prostate.

Hypertrophy
Neoplasm
Cysts
Median bar
Calculi

Ureteral.

Anomalies
- Number
- Origin
- Termination
- Congenital displacements

Congenital valves
Stricture (congenital or acquired)
Ureterocele
Prolapse of the ureter
Atony
Megaloureter
Kinks, torsion, angulation
Retroperitoneal tumors compressing the ureter
Intraperitoneal tumors compressing the ureter
Pregnancy

Tumor, benign or malignant
Inflammation, ureteritis, periureteritis
Calculus
Blood clot

Renal:

Renal anomalies
Aberrant renal vessels
Cysts
Renal ptosis
Calculi
Renal infection (pyelitis, pyelonephritis)
Aneurysm of the renal artery

Pathology: The changes in the renal pelvis and parenchyma are the result of blockage to the normal urinary flow and present the same pattern whether the blockage is partial, intermittent or permanent. In hydronephrosis of a non-infective type, a flattening of the papillae and a gradual thinning of the medulla of the kidney occur. The cortical zone of the kidney gradually undergoes atrophy until only a thin, proliferous wall of the parenchyma exists (FIG. 294).

At first the fluid of the hydronephrosis is composed of urine but as the pressure atrophy of the parenchyma increases the fluid becomes more diluted until it comprises nothing but water and salt. In *infected hydronephrosis* the turbidity of the fluid varies in definite relationship to the number of leukocytes present. Occasionally the fluid is bloody (hematonephrosis); the amount of hemorrhage may be slight or extensive. In a case observed by the author, more than a measured quart of blood was evacuated from a hydronephrotic sac. The opposite kidney developed a reflex anuria following the shock and loss of blood. An emergency nephrostomy, with evacuation of the hydronephrotic sac, followed

by repeated transfusions, sustained life until such a time that nephrectomy could be performed. Section of the removed kidney revealed the wall to be only two millimeters in thickness.

Symptoms: Hydronephrosis does not produce typical symptomatology. This is evidenced by the fact that many cases of hydro-

Fig 294—Hydronephrosis Photograph showing marked sacculation within renal parenchyma
(Temple University Hospital Acc No. 155)

nephrosis are discovered in a routine urological examination, the patient having no symptoms referable to the urinary tract. The symptomatology presented may be that of some intercurrent complication such as infection, calculus or palpable abdominal mass The clinical manifestations of hydronephrosis may be classified under several groupings

1. *Silent:* An extreme hydronephrosis may be apparent without symptomatology and it may be that it is only discovered on complete urological examination. With the general use of excretory urography, such cases are being discovered more frequently than in the past. The frequent finding of a single functioning

Fig. 295—Hydronephrosis. Retrograde pyelogram showing marked hydronephrosis of right kidney.
(Philadelphia General Hospital)

kidney, while its fellow shows no evidence of excretory ability, is not unusual. Following such a roentgenographic finding, retrograde pyelography may reveal the presence of an extensive hydronephrosis (Fig. 295).

2. *Pain and Tumor:* Possibly the most frequent types of symptom complex are those found under this grouping. A dull, aching

sensation may be localized in the costovertebral angle (Fig 296) A tumor may be elicited on palpation in the upper abdominal quadrant or loin In other instances, intermittent attacks of severe colicky pain in the renal area may be described. The pain suggests

Fig 296—Hydronephrosis Retrograde pyelogram showing hydronephrosis produced by extrinsic pressure on the ureter by a uterine fibroid (Philadelphia General Hospital)

the possibility of a ureteral calculus Beginning as a relatively sharp pain in the costovertebral angle, the pain radiates along the course of the ureter Pain may be accompanied by such reflex symptoms as collapse, nausea, vomiting, marked abdominal distention to the point of respiratory embarrassment On occasion, a history will be presented of the presence of a mass which disap

pears following an attack of pain. The palpation of a distinct mass in the upper abdomen is usually possible in the presence of a large hydronephrosis (FIG. 297).

3. *Pyuria:* Infection superimposed upon a hydronephrosis may

Fig. 297—Hydronephrosis. Massive hydronephrotic sac holding 7½ liters of fluid. The sac was so large as to fill the entire abdomen. (Courtesy of Dr. Elmer Hess.)

present a definite pyuria. It is possible to obtain repeated urine specimens that do not reveal the presence of pus cells in a complete closure of the ureter. It is possible in instances of hydronephrosis originating from a partial occlusion of the ureter that following the onset of infection, a definite rise in temperature, chills and malaise may occur, as would be seen in any infective process.

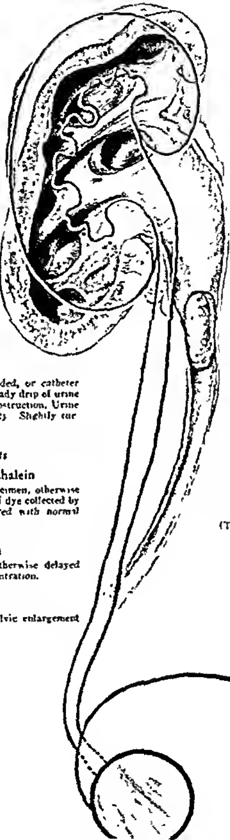
4. *Hematuria:* Hematuria may occasionally be the only suggestive symptom of an existing hydronephrosis. The hematuria is usually intermittent and is the result of congestion of the renal capillaries. Massive hemorrhage may occasionally occur within the hydronephrotic sac, the latter being associated with the symptoms of hemorrhage and shock.

Diagnosis: It is usually possible definitely to establish the presence of a hydronephrosis by our modern methods of examination. Ureteral occlusion may prevent ureteral catheterization and retrograde pyelography. Such occlusions may prevent the diagnosis of a hydronephrosis. There are several factors that should always be considered in making a diagnosis of hydronephrosis.

1. *Clinical history:* A history of pain, its character and distribution should be ascertained. The presence or absence of cloudy urine (pyuria) associated with a history of abdominal tumor should be ascertained. It should be borne in mind that the tumor mass may or may not be palpated. If a mass is present, it may be palpated in the upper abdominal quadrant. In the presence of a ptosis of the kidney, a mass may be palpated at any site within the abdominal cavity. This latter fact is particularly true in the presence of an extremely mobile kidney. Hematuria is not a constant factor. It may never exist. It may be present in microscopic amounts. Seldom is hematuria of gross proportions.

The diagnosis of hydronephrosis requires a consideration of the entire urinary tract (PLATE LXV).

2. *Cystoscopy:* There is nothing pathognomonic of hydronephrosis in the appearance of the bladder. The mode of elimination of indigo-carmine may be suggestive of hydronephrosis. In instances of unilateral hydronephrosis, the normally functioning kidney will excrete the dye in three to five minutes. In the presence of a hydronephrotic kidney, the time of excretion of the dye may be considerably lengthened or entirely absent. The dye may be



Examination

Tumor

In about 25 per cent of instances

Urine

Diminished during attacks. Reflex polyuria following attack of low specific gravity. Microscopically: Negative or occasional pus and red blood cells

Cystoscopic data

Bladder mucosa normal

Meatoscopy

Meatus normal in appearance. Occasional efflux from orifice

Ureteral catheterization

Ureter may be totally occluded, or catheter may pass to renal pelvis. Steady drip of urine from catheter after passing obstruction. Urine usually of low specific gravity. Slightly turbid, few pus cells.

Function tests

Individual Phenolsulfonphthalein

If ureter is occluded, no specimen, otherwise a diminution of the amount of dye collected by ureteral catheter as compared with normal opposite side.

Indigo-carmine elimination

None, if ureter occluded, otherwise delayed appearance time, faint concentration.

X-ray

Pyelogram shows typical pelvic enlargement and blunted calices

Symptoms

Symptoms referred to kidney

Pain

Intermittent attacks of acute pain for months or years. Pain localized to renal region and costovertebral angle. Soreness for a few days after attack

Constitutional symptoms

Nausea, occasional vomiting in some instances. Occasional profuse sweats

Bladder symptoms

Usually none. At times slight vesical irritability.

PLATE LXV.
Hydronephrosis.

(Temple University Hospital Acc. No. 3869.)

seen to be emitted from the ureteral orifice after a prolonged interval The ejection may be a slow trickle of faint concentration

Catheterization of the ureter is of considerable importance in making a diagnosis As the catheter passes an obstruction a con

Fig 298—Hydronephrosis—Hydroureter Retrograde pyelogram showing markedly dilated ureter and renal pelvis caused by stricture of the ureteral orifice. (Temple University Hospital Acc No 74606)

tinuous drip of clear or turbid urine from the catheter is observed It is possible on occasion to evacuate by aspiration through the catheter 100 to 200 or even 300 cubic centimeters of urine from the kidney pelvis (Fig 298) The continuous drip from the ureteral catheter is suggestive of hydronephrosis but may be observed

in hyperactive excretion caused by excitement. A normal kidney function is observed as a series of four to five drops of urine from the ureteral catheter, followed by a pause.

3. *Intravenous urography:* The finding of a nonfunctioning kidney or one only functioning poorly is of significant diagnostic importance. The diagnosis of hydronephrosis can seldom be mistaken if excretory urography reveals that one kidney functions normally and the other shows no function or shows some function and decided loculations. Considerable important diagnostic value may be placed upon the emptying time of a hydronephrosis. Any such roentgenographic finding should be followed by retrograde pyelography.

4. *Retrograde pyelography:* Following introduction of the catheter, the opaque medium should be permitted to enter the hydronephrotic sac by gravity. The burette should be held at a low level to permit the hydronephrotic cavity to fill before exposure of the x-ray plate. The exact capacity of the dilated sac may be clearly demonstrated. It is important to evacuate the opaque medium through the catheter following a retrograde pyelogram. Considerable pain, chills, and fever may develop if the opaque medium is permitted to remain within the renal pelvis. It is also possible to estimate the capacity of the hydronephrosis by measuring the amount of urine or pyelographic fluid withdrawn from the dilated sac through the ureteral catheter.

Hydronephrosis may be bilateral or unilateral. Bilateral hydronephrosis is usually associated with more or less dilatation of the ureters and is frequently associated with atony of the bladder. Bilateral hydronephrosis is usually seen in instances of lower urinary tract obstruction. The obstruction may be of either congenital or acquired origin. Obstruction of congenital origin, such as valves of the posterior urethra or hypertrophy of the verumon-

Fig 299—Hydronephrosis—Extrarenal type Retrograde pyelogram showing dilatation and angulation of ureter.

(Temple University Hospital, Acc No 29488)

tanum may exist Obstructions of acquired origin, such as urethral stricture, hypertrophy of the prostate, vesical calculus, or malignant growths of the bladder are common causes (Fig. 299). Hydronephrosis may also be the result of back pressure occurring in a neurological lesion of the spinal cord, such as syphilis, injuries, or neoplasms of the spinal cord.

Unilateral hydronephrosis is usually the result of some obstructive process lying between the kidney pelvis and the ureteral orifice in the bladder (Fig 300).

Fig. 300—Hydronephrosis. Retrograde pyelogram showing moderate hydronephrosis in the presence of a staghorn calculus.
(Temple University Hospital, Acc No 37786)

Treatment: The treatment of hydronephrosis depends on several factors:

1. Whether hydronephrosis is unilateral or bilateral.
2. The underlying causative factors.
3. The degree to which the hydronephrosis has advanced.
4. The presence or absence of infection and if present, to what extent.
5. Obstruction. Obstruction, with or without infection, is usually the primary cause of hydronephrosis. All therapeutic measures should be directed to ascertaining the cause of the ob-

struction. Infection, although playing an important role, is secondary to obstruction.

All efforts should be made to remove the obstructive uropathy in the lower urinary tract before directing treatment to the upper urinary tract in the treatment of bilateral hydronephrosis. It has been repeatedly demonstrated that following the removal of an obstructive lesion in the lower urinary tract, an apparently hopeless situation of the upper urinary tract will return to a relatively normal condition of moderate or even excellent functioning ability.

In unilateral hydronephrosis, removal of the affected kidney is indicated, if all studies, including pyelography together with the individual function tests, demonstrate irreparable damage to one side. If the damage to the kidney is relatively slight, conservative measures should be instituted rather than sacrifice the kidney. Restoration of function has been repeatedly demonstrated following relief of the obstruction. Nephrectomy is indicated in those instances exhibiting demonstrable neoplasm or tuberculosis. The conservative method of management of a unilateral hydronephrosis is plastic repair of the dilated pelvis by one of the many methods that have been advocated. Catheter drainage may temporarily relieve the acute nature of the symptoms produced by a kinked ureter associated with ptosis of the kidney. The ptosis may be so severe as to necessitate surgical fixation of the kidney for permanent relief of the symptoms.

XXII

NONTUBERCULOUS INFECTIONS OF THE KIDNEY

Infection is one of the most frequent conditions to which the kidney is subjected. It is a subject with which every practitioner of medicine should be acquainted from the standpoint not only of diagnosis but also of treatment. Almost every known type of pathogenic organism has been known to infect the kidneys. The organisms usually observed are rather few. They may be classified as follows:

1. Specific.
2. Nonspecific.

Specific organisms include the tubercle bacillus, *Bacillus proteus,* the gonococcus, *Spirochaeta pallida, Schistosoma haematobium,* echinococcus, and *Actinomyces bovis.*

The nonspecific or pyogenic group includes the *Bacillus coli,* staphylococcus, streptococcus, *Bacillus proteus, Bacillus pyocyaneus, Micrococcus catarrhalis,* pneumococcus, *Micrococcus tetragenous,* and the enterococcus.

From the point of differentiation, the nonspecific organisms may be classified as gram positive and gram negative. The commonest gram negative organisms are the *Bacillus coli communis, Bacillus aurogenes, Bacillus proteus, Bacillus pyocyaneus,* the gonococcus, *Micrococcus catarrhalis, Bacillus typhosus,* and the paratyphoid bacillus. The commonest gram positive organisms are the *Staphylococcus aureus* and *albus,* streptococcus, pneumococcus, *Micrococcus tetragenus,* and enterococcus.

Bacillus coli is the most frequently offending organism. The primary role played by this organism is greater than that of any other organism in the etiology of renal inflammation.

Modes of Bacterial Invasion Infection of the kidney may take place from far distantly removed foci or from an inflammation in contiguous structures It is seldom possible to determine the route of invasion There are three possible routes

1 *Hematogenous route* The hematogenous is the most frequent route by which bacteria enter the kidney and is the result of the direct implantation of bacteria from far distant foci of infection The most frequent organisms responsible are the *Staphylococcus aureus* and the *Bacillus coli* The site of the primary coccal infection may be infected teeth, tonsils, or sinuses, the skin gall bladder, prostate, seminal vesicles or intestines Acute localized suppurative coccal lesions of the skin have been known to be the source of infection in instances of renal carbuncle *Bacillus coli* is possibly the most frequent organism carried by the blood stream to the kidney Bacteremia results not only in the presence of acute inflammatory disease such as colitis or appendicitis but also following the passage of urethral instruments in the presence of an infection in the lower urinary tract Coccal infections are usually more serious the bacteremia caused by the *Bacillus coli* is usually transitory unless unusually virulent

2 *Urogenous route* The question of ascending infections within the lumen of the ureter is a very debatable one and one that is refuted by many The occurrence of ascending infection has been proven by many investigators Ascending renal infection is undoubtedly due to a vesicoureteral reflux The occurrence of the latter is the result of an incompetent ureterovesical sphincter in the presence of some type of obstructive uropathy Infection of the kidney by the urogenous route is most apt to occur in the presence of neurogenic dysfunction of the bladder, a large vesical calculus in many instances of prostatic enlargement or obstructive uropathy in the lower urinary tract

3 *Lymphogenous route* The idea that infection is carried to

the kidney by the lymphogenous route is a very disputed question. It is believed that infection traversing primarily the lower lymphatics is carried through the thoracic duct into the general circulation and then to the kidney rather than directly up the lymphatics surrounding the individual ureter. Considerable experimentation has been done but as yet there is no uniformity of opinion as to the direct transmission of bacteria from the lower urinary tract to the kidney by way of the lymphatics.

The accessory factors favoring invasion of the kidney by bacteria are obstruction and trauma.

Obstruction: Obstruction may be congenital or acquired and may be at any site in the urinary tract. Regardless of its site, obstruction not only interferes with normal drainage but predisposes to infection. The same factors are present regardless of whether the urinary obstruction is acute or chronic, congenital or acquired. The stagnation of urine behind or above the obstruction invariably traumatizes the delicate tissues by distending them. The blood supply is disturbed by the distention and the urine becomes an irritant to the traumatized mucosa. It is in this way that a vicious circle is established and as one factor continues or increases, so does the other. Tissue destruction follows implantation of bacteria as the normal resistance of the mucosa is lowered. Once infection has been established the same factors which predisposed the tissues to infection hamper or even prevent a cure until these factors are eliminated. In renal infection it is imperative to establish the presence or absence of any obstructive process before attempting treatment.

Trauma: Trauma to the kidney, regardless of its nature, may be an accessory factor in the production of a renal infection. Not only may the traumatized tissue be the result of some obstructive lesion but direct trauma such as a blow to the kidney or the passage of a calculus may prove to be a definite etiological factor. Certain drugs

or chemical toxins such as mercury, bismuth, arsenic, or other heavy metals produce indirect trauma as they are eliminated by the kidney. Bacterial toxins present in glomerulonephritis may likewise cause trauma to the kidney.

Pathology: The pathology of renal infection or renal inflammation of the surgical infections of the kidney must be differentiated from the pathology of noninfectious, nonsuppurative medical conditions of the kidney. As has been previously pointed out, the noninfectious lesions of the kidney are considered to be nephritis and nephrosis and usually come directly under the observation of the internist. This fact should never be overlooked, that though a kidney may exhibit noninfectious lesions it may also be attacked simultaneously by suppurative lesions. It remains for the urologist to make a differential diagnosis and to outline the management of the disease.

Suppuration or acute pyogenic inflammation of the kidney is predominantly caused by the *Staphylococcus aureus,* although the *Staphylococcus albus* and *Streptococcus pyogenes* or *hemolyticus* may be the causative cocci. *Bacillus coli* is the most commonly found bacillus and its presence usually occurs with obstructive uropathy. The staphylococcus is implanted most frequently *within the kidney by the hematogenous route. The pyogenic* cocci, that is, the staphylococcus or streptococcus usually lodge in the cortex and especially in the glomeruli. Such an implantation is followed by a rapid suppuration of the kidney structure although only a few pus cells and few red blood cells are found in the urine. The urine is usually alkaline due to the ammòniuric action of these organisms. The *Bacillus coli* usually invades the mucous membrane of the pelvis to invade later the cortex of the kidney. Such an infection is usually associated with enormous numbers of pus cells in the presence of a strongly acid urine. The ascending infection by the *Bacillus coli* is much less rapid and may progress

slowly over a period of years. Infection by the staphylococcus is usually quite rapid running a typically febrile course of increasing intensity.

DIFFUSE SUPPURATIVE NEPHRITIS

(pyemic kidney)

Diffuse suppurative nephritis is also known as pyemic kidney. The lesions found within the kidney itself are only a part of a generalized pyemia. The condition always occurs in the presence of some distant but acute suppurative process such as osteomyelitis, carbuncle of the neck, furunculosis, an ulcerative endocarditis, or puerperal sepsis. Diffuse suppurative nephritis may also occur in association with any other condition in which there is a massive, increasing, uncontrolled infection. The causative organism is usually the *Staphylococcus aureus* although the *Streptococcus hemolyticus* may occasionally be the offending organism.

Pathology: Both kidneys are involved and present a swollen and congested appearance on examination. The capsule is tightly stretched over the surface of the kidney and when removed, presents a surface that is studded with small, minute, yellow abscesses which are slightly raised and present a hemorrhagic margin. The cut surface of the kidney presents minute, yellow abscesses which are slightly raised and present a hemorrhagic margin. These abscesses are minute, multiple, and circular in the cortex, while in the medulla they take the form of linear streaks lying between the straight tubules. Microscopically, the minute abscesses consist of polymorphonuclear leukocytes and masses of cocci. The capillaries of the glomeruli show only a slight inflammatory reaction although they contain masses of the microorganisms.

In spite of the massive invasion of bacteria and formation of multiple abscesses, the urine shows very few pus cells

The clinical picture presented is that of an acute, intense febrile reaction associated with generalized septicemia Since the invasion of the kidney is so rapid and termination so universally fatal little destruction of the kidney takes place

FOCAL SUPPURATIVE PYELONEPHRITIS

Focal suppurative pyelonephritis may be considered as those pyogenic forms of renal infection that are neither the result of the staphylococcus focal infection of the cortex nor the specific types of suppurative nephritis Focal suppurative pyelonephritis is generally induced by the *Bacillus coli* although the staphylococcus or a mixed infection may be found Both kidneys may be affected but it is seldom that both are equally involved The condition is commonest following a cystitis in the presence of obstructive uropathy of the lower urinary tract such as stricture, prostatic hypertrophy or vesical calculus The condition may be of hematogenous origin the pelvis of the kidney being secondarily infected Focal suppurative pyelonephritis may be considered under five headings each a stepping stone to the other

1 Pyelitis
2 Pyelonephritis
3 Atrophic pyelonephritis
4 Infected hydronephrosis
5 Pyonephrosis

1 Pyelitis Pyelitis is an inflammation of the kidney pelvis Some investigators consider pyelitis to be a clinical entity in itself They consider that the infection is always limited to the mucosa of the renal pelvis tends to remain localized in the acute stage and may continue through subacute stages to become chronic in some

instances. It is believed by the author that pyelitis, *per se,* is the mildest form of renal infection in which an infection beginning in the kidney pelvis spreads rather quickly into the parenchyma of the kidney; that pyelitis, if permitted to progress unhampered and untreated, will progress successively through the stages of pyelonephritis, infected hydronephrosis, and ultimately pyonephrosis. Pyelitis is not an isolated condition but rather the first stage of a continuously advancing destructive inflammatory process. The infection may arise from one of two routes, the hematogenous or the ascending route. The ascending route from the bladder is the commoner. In the hematogenous infection the parenchyma is first involved, the pelvis secondarily. In the ascending route the infection first attacks the mucosa of the pelvis then spreads to the parenchyma of the kidney.

Bacillus coli is the most frequently offending organism. The organisms undoubtedly ascend in the wall of the ureter or by regurgitant action through a dilated ureter directly to the kidney pelvis. The regurgitant action occurs through an incompetent ureterovesical valve.

The clinical picture presented is similar whether invasion occurs by the hematogenous or ascending route after a relative period of time, 48 to 72 hours.

2. Pyelonephritis: Pyelonephritis may be acute or chronic (FIG. 301). In the acute hematogenous infections the earliest changes take place within the renal cortex. Examination of the cut surface of the kidney reveals it to be studded with pin-head sized hemorrhages or by small nodules elevated above the surface of the adjacent parenchyma. These nodules have a yellowish center surrounded by an intensely red areola. As the infection spreads into the pelvis the mucous membrane presents multiple minute hemorrhagic areas or is diffusely red and edematous.

The cut surface of the kidney presents wedge-shaped areas, dark

Fig 301—Pyelonephritis with an associated calculus within the renal pelvis
(Temple University Hospital)

red in color mottled with areas of suppuration These wedge shaped areas are due to the manner of spread of the infection from the pelvis toward the cortex of the kidney along the line of the renal tubules Such an appearance resembles a renal infarct and is known as Brewer s septic infarct These areas may undergo abscess formation In the acute ascending infection the pelvis is the first to become inflamed The mucous membrane becomes red and edematous or shows multiple minute hemorrhagic areas Secondarily the renal cortex presents a picture of small multiple

Fig. 302—*Pyelonephritis.* Intravenous urogram. Note blunting of calices of right kidney. Left kidney normal.
(Temple University Hospital, Acc No 30053.)

hemorrhagic areas or minute abscesses. It is possible for these abscesses near the periphery of the kidney to rupture into the perirenal tissue with the formation of perinephritic abscess or to become a localized abscess known as carbuncle of the kidney.

Acute pyelonephritis may be of hematogenous origin primarily; later the infection invades the pelvic mucosa. More frequently the bacterial invasion of the parenchyma is preceded by a pyelitis of ascending origin. The infection spreads secondarily into the parenchyma (FIG. 302).

Microscopically, small abscesses may be seen lying in the renal

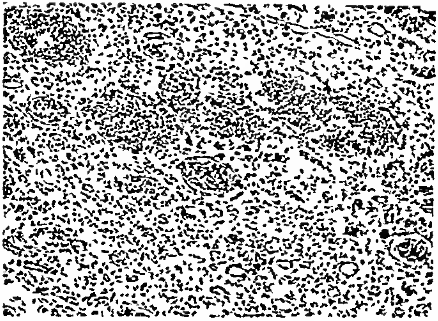


Fig 303—Kidney—pyelonephritis Low-power photomicrograph showing several collecting tubules filled with purulent exudate—also diffuse interstitial suppurative infiltration.
(Courtesy of Dr Lawrence W Smith and Dr Edwin S Gault)

stroma between the tubules and glomeruli as well as between the glomeruli and pyramids The abscesses tend to coalesce, ultimately causing marked destruction of the kidney This is particularly evident in the presence of renal calculus when obstruction is added to the infection present The inflammatory changes of the mucosa of the pelvis are those of any mucous membrane when subjected to infection (Fig. 303). In the presence of a chronic infection of long standing, the mucosa may present minute cysts (pyelitis cystica), luxuriant granulations (pyelitis granulosa) the presence of lymph follicles (pyelitis follicularis), soft patches (malakoplakia), or dermal-like thickening (leukoplakia) Each of these conditions is discussed elsewhere.

Ascending or descending infections of the kidney cannot be differentiated once the infection has been established Mixed

Fig. 304—Pyelonephritis. Retrograde pyelogram showing blunting of calices and sharp angulation of ureter.
(Temple University Hospital, Acc. No 46678.)

infection of both cocci and bacilli frequently exists. As the original bacteriological invaders lose their virulence secondary bacteria are implanted. The lesions of the kidney may heal spontaneously and quickly, or may rapidly develop into abscesses or may become chronic (Fig. 304). If the lesions become chronic and are untreated they progress into atrophic pyelonephritis or pyonephrosis.

3. Atrophic Pyelonephritis (*Chronic sclerosing pyelonephritis*): Atrophic pyelonephritis is usually the result of long-standing chronic infection with multiple abscess formation. Pyelectasis is

absent which shows that obstruction is not one of the basic causative factors The kidney is markedly contracted, its capsule is thickened and adherent, and when removed shows on the surface many irregular depressed white scars to which the capsule is adherent The kidney of atrophic pyelonephritis is smaller than normal being reduced to one quarter or one third of its original size The condition is usually unilateral The atrophic kidney is usually embedded in densely adherent perirenal tissues so that at operation the position of the kidney may be difficult to locate Once the kidney is located mobilization may be so difficult as to be impossible Atrophic pyelonephritis is to be differentiated from primary atrophy, infantile kidney and the sclerotic kidney of Bright s disease The atrophy of pyelonephritis may be so diffuse as to resemble primary atrophy, or so circumscribed as to resemble an infarct These differences of appearance depend entirely on the healing processes that follow a diffuse chronic pyelonephritis or localized cortical abscess formation

4 Infected Hydronephrosis· Pyelectasis or hydronephrosis tends to follow any variety of obstructive uropathy of the urethra, vesical neck, bladder or ureter Infection is sooner or later superimposed upon hydronephrotic atrophy Infected hydronephrosis begins as a diffuse pyelonephritis with dilatation of the pelvis and calices As the condition advances secondary pressure atrophy occurs resulting in cessation of excretory function In infected hydronephrosis the parenchyma is compressed but not destroyed, the abolished function resulting from pressure atrophy Throughout this compressed parenchyma, a diffuse pyelonephritis may be seen As a result of the long-continued process pyonephrosis develops

5 Pyonephrosis Pyonephrosis is the end stage of destruction of renal tissue (*Plate LXVI*) There is a purulent inflammation of the kidney with pouching and dilatation of the pelvis and calices

PLATE LXVI—Ribbon of pus exuding from left ureter in the presence of pyonephrosis.

associated with marked destruction of the renal parenchyma Such destruction leaves the kidney a flabby, functionless bag containing thick creamy pus (Fig 305) The kidney is greatly enlarged and

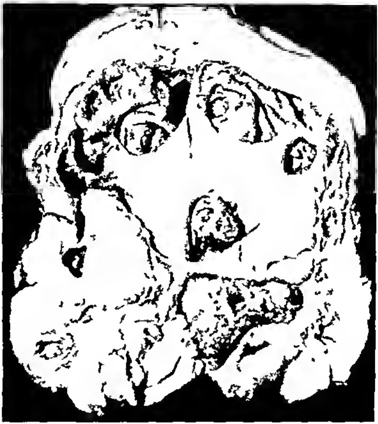
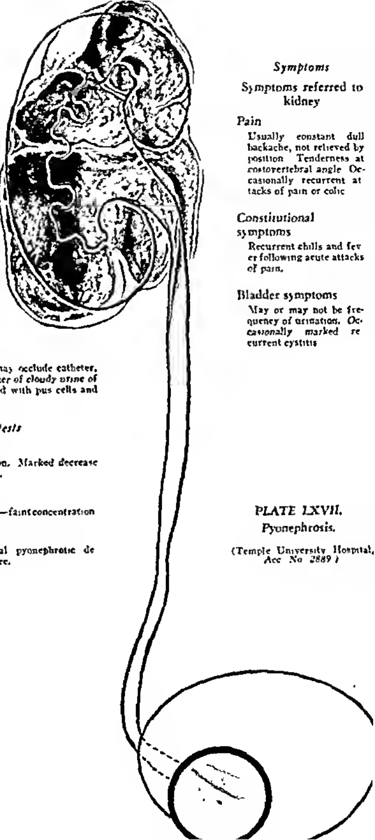


Fig 305—Calculous pyonephrosis Marked distention of pelves with almost complete destruction of renal parenchyma a mere shell of kidney substance remaining.
(Temple University Hospital Acc No 2399)

markedly lobulated A series of communicating cavities representing the dilated calices connects with an enormously dilated renal pelvis The kidney has been converted into a functionless infected dilated sac Removal is the only treatment (PLATE LXVII)

Symptoms The symptoms of pyelonephritis are generally of



Examination

Tumor

Usually a tender mass in renal region.

Urine

Loaded with pus cells and bacteria Hematuria is the exception

Cystoscopic data

Bladder mucosa somewhat congested, ureteral orifice usually inflamed.

Meatoscopy

Congestion may be *localized or part of a* moderate cystitis.

Ureteral catheterization

Pus and cellular debris may occlude catheter, *or steady drip from* catheter *of cloudy urine* of low specific gravity loaded with pus cells and cellular debris.

Function tests

Phenolsulfonphthalein

Markedly delayed function. Marked decrease in percentage elimination.

Indigo-carmine

Delayed appearance time—faint concentration

X-ray

Pyelogram shows typical pyonephrotic deformity in varying degree.

Symptoms

Symptoms referred to kidney

Pain

Usually constant dull backache, not relieved by position Tenderness at costovertebral angle Occasionally recurrent attacks of pain or colic

Constitutional symptoms

Recurrent chills and fever following acute attacks of pain.

Bladder symptoms

May or may not be frequency of urination. *Occasionally marked* recurrent cystitis

PLATE LXVII.

Pyonephrosis.

(Temple University Hospital, Acc No 2889)

sudden onset (Fig 306) The onset is usually marked by chills and fever The condition may be mistaken for a systemic infection, such as influenza Definite urinary symptoms such as frequency, urgency, dysuria, and pyuria develop after a few hours

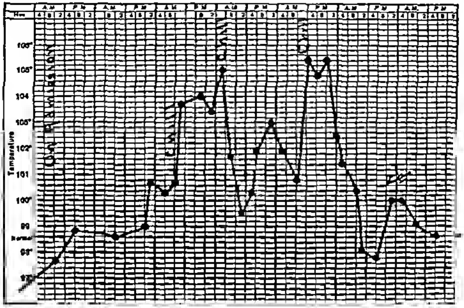


Fig 306—Pyelonephritis Temperature chart showing inception and progress of *acute pyelonephritis during treatment.*

These urinary symptoms are usually accompanied by pain and tenderness localized in the loin and particularly at the costovertebral angle Gastrointestinal symptoms nausea, vomiting, or even a mild diarrhea generalized abdominal pain, and distention may occur Leukocytosis is usually present

Urine Of utmost importance is a complete and accurate survey of the urine Pyuria is evident (Fig 307) The urine is usually turbid and acid in reaction Acute pyelonephritis cannot exist without pyuria if the ureter is normally patulous Microscopic examination of the turbid urine reveals pus cells red blood cells

cellular debris, and organisms. Normal urine findings will rule out the possibility of acute hematogenous pyelonephritis or cortical abscess of staphylococcic origin. Urine culture will yield a report of much importance. The offending organism may be

Fig 307—Pyonephrosis. Retrograde pyelogram showing marked dilatation of pelvis and erosion of calices.
(Temple University Hospital, Acc No 30847.)

grown and studied. Subsequent culture will reveal any changes or addition or other organisms. Although *Bacillus coli* is the most frequently offending organism, mixed infections occur very frequently.

The acute symptoms usually subside within two weeks. The

symptoms may disappear in some instances without leaving a trace of infection Persistent evidence of infection following an acute attack may be due to preexistent lesions or that the infection has become subacute or chronic

Albuminuria Albuminuria is present and results from the abnormal constituents in the urine Albuminuria is always present whenever hematuria occurs In acute pyelitis visible hematuria occurs frequently Microscopic hematuria occurs in almost every instance of acute pyelitis Casts are absent in cases uncomplicated by nephritis

Diagnosis *Renal function tests* Urinary output is diminished there is considerable loss of renal function as shown by phenolsulfonphthalein elimination The diminution of phthalein output may be unilateral or bilateral depending on whether a unilateral or bilateral infection is present In spite of this loss of renal function there is little or no nonprotein nitrogen retention in the blood This fact may be explained by the limited involvement of the glomeruli in contrast to the high nitrogen retention in acute diffuse glomerulonephritis in which all the glomeruli are involved

Cystoscopy Cystoscopic examination should be done in all cases of pyelitis and pyelonephritis except in those instances manifesting hyperacute symptoms or in those instances where cystoscopy is definitely contraindicated Cystoscopy should not be attempted in debilitated individuals with evident but yet borderline cases of uremia or in evident cardiac impairment An acute severe cystitis is a definite contraindication to cystoscopy

Acute pyelitis and pyelonephritis are usually associated with an inflammatory reaction of the bladder mucosa In some instances only a hyperemic blush around one or both ureteral orifices may be noted The blood vessels of the mucosa are promi

nently engorged and appear to be increased in number, suggestive of congestion rather than inflammation. Usually some degree of cystitis is present. The cystitis presents the appearance of a localized type but occasionally the inflammation may be generalized. The mucosa covering the trigone is very apt to be inflamed and edematous. Considerable amounts of free floating clumps of pus and debris may be seen in the distending medium. Masses of mucopus may be lightly attached to the mucosa and may be readily washed away by the distending fluid. Occasionally it is possible to observe an efflux of turbid urine ejected from the ureteral orifice. This turbid urine is constantly evacuated into the bladder. The mucopus and debris from the inflamed bladder mucosa may necessitate continued irrigation before clarity of the distending medium is attained. Accurate cystoscopic visualization is impossible in the presence of a markedly turbid medium.

Ureteral catheterization: Much important clinical data may be obtained by bilateral catheterization of the ureters and collection of individual specimens. Harm may be done by indiscriminate catheterization of a ureter to a normal renal pelvis in the presence of cystitis unless definite precautionary measures are taken. Ureteral catheterization is essential and should be done if an accurate diagnosis is to be made. When ureteral catheterization is undertaken it should be done correctly or not at all. (See technic of ureteral catheterization and collection of ureteral specimens.)

INFORMATION GAINED BY URETERAL CATHERIZATION:

1. Knowledge of the patulousness of the ureter.
2. Whether the infection is unilateral or bilateral.
3. The nature and characteristics of offending organisms.
4. The degree of destruction or functional impairment of both kidneys.

The blocking of one ureter presents a clinical picture that is of great clinical importance, particularly if the normally functioning kidney is infected It is important to know that both ureters are open and functioning as well as the functioning ability of both kidneys

The urine withdrawn from the infected side is hazy or cloudy It is not heavily purulent except in the presence of suppurative destruction of the kidney In bilateral infections the cloudiness of the two specimens is not usually equal The urine from one

Fig 308—Actual photograph showing ejection of indigo-carmine from ureteral orifice

kidney is usually more purulent than that from the other Urine specimens for culture should be taken from both kidneys By such procedure unilateral or bilateral involvement may be ascertained The identity of the offending organism may be learned and the bacteriological growth of both sides compared

Differential functional tests Indigo carmine or phenolsulfonphthalein may be used With the use of the latter it is possible to ascertain a percentage of elimination from both kidneys for comparison The excretion of the dye is slightly delayed as well as diminished in acute pyelitis or pyelonephritis The appearance of the dye after intravenous injection is normally from three to five minutes (Fig 308) A delay varying from six to eight minutes

may be noted in the presence of infection. The normal percentage elimination of phenolsulfonphthalein in a 15-minute period is 15 to 17 per cent. In acute pyelitis or pyelonephritis a percentage elimination of seven to ten per cent is usual.

Fig. 309—Pyonephrosis. Intravenous urogram showing calcification within a pyonephrotic sac.
(Temple University Hospital, Acc. No. 25339.)

Intravenous urography: Excretory pyelography is of material aid and may be used at any stage of the infection (Fig. 309). Although a normal roentgenogram does not exclude inflammation, a grossly abnormal pyelogram is of great aid (Fig. 310). Gross pathology or congenital anomalies may be readily observed. The

finding of either may be a guide for further investigation and study. Excretory urography should always precede cystoscopic examination, ureteral catheterization, or retrograde pyelography. Excretory urography should be considered as a guide for

Fig 310—Pyonephrosis. Retrograde pyelogram showing marked dilatation of the renal pelvis and destruction of the calices.
(Temple University Hospital, Acc No 26299)

position and excreting ability of both kidneys Obstructive lesions accompanied by distention of the ureter and kidney pelvis, or a functionless kidney never before suspected, may be brought to light. It is generally conceded that excretory urography in pyelonephritis rarely permits adequate visualization of the full extent

of destruction caused by the infection. The most expert interpretation will fall amiss of the characteristic ectasis and blunting of the minor calices (Fig. 311). These characteristic deformities of the calices and ureters are more accurately visualized by retro

Fig. 311—Pyelonephritis. Retrograde pyelogram showing blunting of calices. (Temple University Hospital, Acc. No. 41644.)

grade pyelography which should be done following the preliminary excretory urogram.

Retrograde pyelography is contraindicated in the presence of fever and localized tenderness. Such a study should be done after the acute phase of the infection has subsided.

Prognosis: The prognosis of acute pyelonephritis depends pri-

marily on the type and the virulence of the offending organisms It is undoubtedly true that acute pyelonephritis would never occur in the absence of certain factors favoring the ingress of bacteria within the kidney Beyond question the most favorable predominating factor is urinary stasis and the tissue trauma accompanying such stasis In the majority of instances stasis predisposes to infection Once an infection is established it will continue until the obstruction is relieved or corrected This is proven in instances of acute but mild attacks of pyelonephritis Spontaneous healing occurs without permanent impairment of function following the relief of obstruction Subsequent urine cultures will be found negative This fact is also proven when improvement in the clinical symptoms is gained by temporary measures of treatment Recurrent attacks of pyelonephritis are common Complete eradication of infection is possible only in a urinary tract that is structurally and functionally normal Persistent gross pyuria following an attack of acute pyelonephritis with mild recurring symptoms is indicative of structural damage of the urinary tract with stasis as an underlying and predominate causative factor Not that all infections of the upper urinary tract will heal spontaneously after removal of the obstructive lesion but as long as stasis is present, infection, when once established, will continue

Treatment Eradication of the underlying and causative factors is the primary purpose of treatment Formerly the symptoms of acute pyelonephritis were *treated as they became apparent It is* an established fact that elimination of the cause is generally followed by spontaneous subsidence of the infection This statement is true provided the effects of infection are not too far advanced No intelligent outline of treatment can be inaugurated unless based on an accurate diagnosis In the acute stage of infec

tion it is impossible to establish such an accurate diagnosis even with our modern methods of investigation because such diagnostic procedures are contraindicated. Under such circumstances, sound logic and accurate interpretation of the presented facts are important.

The points to be considered are:

1. The nature and bacteriological characteristics of the invading organism or organisms.
2. The mode of entry of the offending organism whether hematogenous or by the ascending route.
3. The presence of obstructive factors producing stasis, either of congenital origin or of a pathological entity.
4. The degree of destruction or loss of functional activity of one or both kidneys.

A correlation of the facts presented will enable intelligent treatment to be instituted. Basically the principles of the treatment of ascending acute pyelonephritis are: (1) Systemic and dietary measures; (2) maintenance of unfavorable medium for the growth of bacteria; (3) drainage.

1. Systemic and Dietary Measures: The patient should be confined to bed. The bowels should be kept open by a saline purge repeated whenever necessary. A nourishing but bland liquid diet should be maintained. The daily intake of fluids should be raised, double the normal amount of intake if possible. The normal output of urine is 1500 to 1800 cc. (50 to 60 ounces). By increasing the intake the output is raised to 3000 to 3600 cc. (100 to 120 ounces). Nausea or vomiting may be induced by the ingestion of so great a quantity of fluid. These distressing symptoms may be eliminated by the daily intravenous administration of 1000 cc. of a five per cent glucose solution in normal saline solution. Whole blood transfusions are usually not indicated although adminis-

tration of properly typed whole blood will aid materially in the management of a hyperacute infection

2 Maintenance of Unfavorable Medium for the Growth of Bacteria The production of an unfavorable medium for bacterial growth is best maintained by alkalinization of the urine The usual causative organism is the *Bacillus coli* which is known to

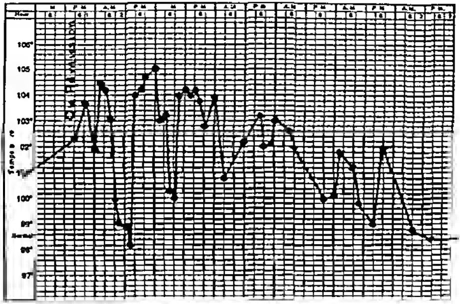


Fig 312—Pyelonephritis Temperature chart showing regression of acute pyelonephritis Therapeutically alkalies in massive doses were administered.

thrive best in an acid medium By alkalinization of the urine an unfavorable medium is created Alkalinization is best maintained by the oral administration of potassium citrate 4 Gm (60 grains) and sodium bicarbonate 2 Gm (30 grains) every three hours until the temperature falls to normal (FIG 312) This drop in temperature usually occurs in three to four days Under such dosage the *p*H of the urine rises to six or above Such a level may be maintained by the continued administration of alkalies in

smaller doses. Usually potassium citrate, 2 Gm. (30 grains) and sodium bicarbonate, 1 Gm. (15 grains), administered three times a day, is sufficient to maintain the desired *p*H level.

3. Drainage: Continuous drainage by means of a ureteral catheter is most effective in the presence of obstruction. The ureteral catheter must pass the obstruction to be of any benefit. The catheter must be introduced to the level of the renal pelvis and permitted to remain *in situ* from three to five days. It is seldom necessary to repeat ureteral catheterization or sustain ureteral drainage for a longer period. Instillation of bactericidal solutions through the indwelling catheter is contraindicated in the acute phase of infection.

Following the onset of acute pyelonephritis it is doubtful if the oral administration of urinary antiseptics is beneficial. Urinary antiseptics are irritants to the acutely inflamed renal tissues. Although numerous urinary antiseptics have been advanced none has proven itself to be universally successful. Regardless of its apparent efficiency in a test tube none will eradicate an infection of the urinary tract in the presence of stasis or obstruction. In many instances the symptoms will be intensified by such antiseptics which possess irritating qualities when administered in the presence of urinary obstruction. As a routine therapeutic measure urinary antiseptics accomplish less than the alkaline diuretic treatment. Furthermore, such drugs are poorly tolerated by the usually irritable gastrointestinal tract. Chemotherapy is not advised during the acute stage of pyelonephritis.

The theory of alternate alkalinization and acidification is widely accepted and widely used. Such therapy is basically unjustifiable particularly when the causative organism is the *Bacillus coli*. This organism is favored in its growth by an acid urine. Why then should the medium be made unfavorable for its growth by alkalinization and then made favorable by acidification (FIG. 313)?

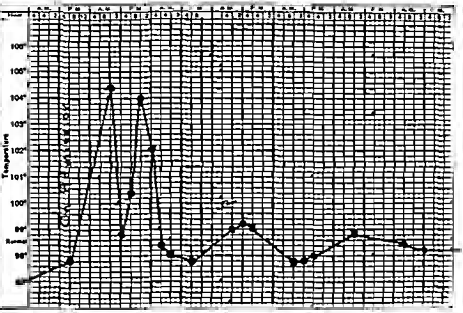


Fig 313—Pyelonephritis Temperature chart showing rapid regression of acute pyelonephritis following administration of alkalies in massive doses

CHRONIC INFECTIONS OF THE KIDNEY

Chronic pyelonephritis may persist for years following an acute attack of the infection The chronicity is favored by stasis caused by obstructive uropathy or an anomaly of the urinary tract In this type of chronic infection attention must be directed to the localization and elimination of the obstructive mechanism producing the stasis Elimination of the obstruction and the production of normal adequate drainage will tend to facilitate the eradication of the infection Chronic infection may persist for years resisting all attempts to eradicate it unless obstruction is eliminated with the production of normal drainage The roentgenogram of the kidney pelves and calices shows distinct blunting and distortion These changes are the result of stasis and infection

Chronic pyelonephritis may arise from various primary sources It may follow an acute pyelonephritis or may develop insidiously

from a focus of infection in the skin, teeth, tonsils, nasal sinuses, appendix, gallbladder, prostate in the male, or cervix uteri in the female.

Pathology: The parenchymal lesions vary from moderate congestion and cellular infiltration to chronic suppuration and sclerosis. The renal pelvis shows some dilatation and the walls are thickened. Dense adhesions to the peripelvic fat are usually present. The changes in the mucosa of the pelvis vary from a red, congested, and edematous appearance to one in which the walls are covered with a fibrinoplastic exudate or to definite ulceration of the mucosa. In a certain percentage of instances which have shown a long-protracted chronic infection the mucosa of the pelvis may exhibit unusual appearances.

(*a*) *Leukoplakia:* The clinical significance of this metaplastic change of the mucosa is uncertain. Long-protracted chronic infection is the only known cause. There are several theories as to the underlying factors and significance of the condition but none has been proven. Kretschmer, in a survey of 13 cases, was unable to demonstrate any other pathology in the genitourinary tract. One very important clinical factor is evident. The condition is incurable and is considered by some investigators to be the forerunner of squamous-cell carcinoma. When present in the renal pelvis it will be usually observed that similar changes have taken place in the bladder and ureter (Fig. 314). Such changes may be observed in the bladder alone, the ureters and renal pelves being free from this metaplastic change. Patch, in an analysis of 152 cases, found leukoplakia to occur 110 times in the bladder, 36 times in the renal pelves, and only six times in the ureter.

(*b*) *Pyelitis cystica:* Pyelitis cystica is a form of pathological change occurring in the presence of chronic infection. The condition is a metaplasia of the epithelial lining of the renal pelvis.

Pyelitis cystica is characterized by the formation of small cysts in or beneath the mucosa. Similar changes may be noted in the bladder and in the ureter. These cystic changes may become so extensive as to present a characteristic yet bizarre appearance on a ureteropyelogram. Hinman describes this condition as . . . 'Characterized by downgrowths of the epithelium which in some

Fig 314—Leukoplakia of the kidney pelvis.
(Courtesy of Dr A J Scholl and *Journal of Urology*, 1940, 44 759)

areas have become separated entirely from the surface epithelium to form cysts The cysts are lined by a single or double layer of epithelium, are filled with a clear fluid resembling colloid and lie directly beneath the surface. There are collections of round cells scattered throughout the mucosa but no true lymph follicles have been found."

The presence of these cysts is of clinical importance because of the persistence of continued infection that resists all treatment.

(c) *Pyelitis granulosa:* Pyelitis granulosa is a lesion of the pelvic mucosa that is observed in the presence of chronic infection. This condition is characterized by granulations and circumscribed lymphoid follicles. These small papillary areas are composed of small blood vessels supported in loose areolar tissue. Due to their vascularity they are responsible for recurrent or persistent hematuria in chronic renal infections.

Symptoms and Diagnosis: The classical symptom of chronic pyelonephritis is pyuria. Varying numbers of pus cells in the urine are characteristic. The number of pus cells varies from day to day. One day the urine may contain only a few pus cells while innumerable pus cells may be found on the next day. The number of pus cells will be increased in the event of an acute recurrent attack with associated pain in the loin, chills, and fever. The number will be diminished as the acute nature of the attack subsides. Intermittent appearance of painless hematuria (gross or microscopic) is common. The hematuria is seldom of alarming proportions and usually subsides as quickly as it appears. Repeated examinations of the urine show that the pyuria exists. Symptoms referable to the bladder are usually mild and transient although symptoms of a chronic protracted cystitis may be observed. Marked frequency and urgency of urination may be the dominating symptoms. The bladder symptoms may be so predominate as to overshadow the infection of the kidney.

Physical examination reveals few positive findings. Seldom are any important or characteristic data obtained by physical examination. A slight tenderness at the costovertebral angle may be the only finding of any significance.

Urine studies: Repeated urinalysis will reveal the progressive or regressive activity of the chronic infection. Although the urine varies in content from day to day it is possible to estimate

the changes that are present or that have taken place over a period of time

Urine cultures will disclose the causative organism Urinary tuberculosis should be excluded in any instance of persistent pyuria The urine should be subjected to routine culture as well as a culture for the tubercle bacillus A portion of centrifuged sediment should be stained for the demonstration of the tubercle bacillus

Renal function, as estimated by phenolsulfonphthalein, is usually slightly reduced but is never reduced to extreme degrees

Cystoscopic examination There are no pathognomonic vesical lesions of chronic pyelonephritis The bladder, in the presence of a chronic pyelonephritis, presents a cystoscopic picture that varies from a mild, patchy, congestive cystitis to a marked interstitial cystitis The area immediately surrounding the ureteral orifices may present a red congested appearance or may show areas of bullous edema In certain instances of long protracted infection, cystitis glandularis, cystitis cystica, or patches of leukoplakia may be observed

As the distending medium fills the bladder, small flakes of mucopus may be dislodged from the inflamed mucosa and float aimlessly in the fluid The appearance time and concentration of indigo carmine intravenously administered may be found to be normal but is usually slightly delayed The degree of concentration depends on the extent of destruction resulting from the infection

Ureteral catheterization should always be done An individual specimen for analysis and culture should be taken from each ureter Care should be taken in the collection of the specimen for culture so that the ureteral catheter is not contaminated by infected fluid within the bladder A false report of the culture is certain to follow

Intravenous urography: Although intravenous urography should be done routinely as a preliminary study it frequently fails to outline the fine details of the minor calices as accurately as retrograde pyelography. Considerable information is to be gained by intravenous urography in the presence of a chronic infection. Such vital data as the functional ability, the contour, the position of the kidneys as well as the presence or absence of stasis, may be estimated. Simultaneous bilateral retrograde pyelography is never advised. It should be remembered that a negative appearing pyelogram does not exclude the presence of pyelonephritis. The typical roentgenographic finding of an irregular, moth-eaten or hazy outline of the calices is significant of infection of the kidney. An increase of this irregularity, with dilatation and blunting of the calices, is definite evidence of destructive renal infection. Small multiple filling defects of pyelitis cystica may be observed on the walls of the renal pelves. These defects may be confused with or appear as air bubbles in the midst of the pyelographic medium.

Treatment: The treatment of chronic pyelonephritis comprises the utilization of numerous therapeutic factors. All these measures depend upon the condition found on examination of each individual. Regardless of an accurate diagnosis and a thorough estimation of the underlying factors and the nature of the invading organism, some instances of chronic renal infection will continue in spite of all methods of treatment. The best results are obtained by elimination of the underlying factors of obstruction and stasis with attention secondarily directed to the offending organisms. Removal of the obstruction and the production of adequate drainage are frequently followed by a rather prompt disappearance of the infection. In those instances where the infection continues, various modes or methods of treatment may be

necessary before the infection is eliminated. These different measures are:

A. General hygienic measures
B. Medication—oral administration
 Urinary antiseptics
 Urinary acidifyers and alkalinizers
 Chemotherapy
 Penicillin
 Ketogenic diet
 Acid ash diet
 High alkaline-ash diet
C. Vaccine therapy—bacteriophage
D. The indwelling ureteral catheter
 Ureteral dilatation
 Pelvic lavage
E. Surgery

A. *General Measures:* The patient's general physical condition should be maintained at as high a level as possible. Rest is essential. A well-balanced diet with a high caloric value is indicated. Bowel elimination should be adequate and regular. Alcohol in any form or amount is contraindicated

B. *Medication: Urinary antiseptics* are usually palliative. Urinary antiseptics may control but seldom eradicate chronic renal infection The choice of the drug to be employed depends on the nature of the offending organism as well as the tolerance of the particular drug by the patient. Many urinary antiseptics have been compounded. The list of these compounds is long but few have withstood clinical trial Some of these drugs have been in favor for a short time, have been found wanting in efficient action and have been discarded Possibly the most efficient in action and one that has withstood the crucial test of clinical use is methenamine (urotropin). Introduced in 1894 by Nicolaier, it is still one

of the most dependable and widely used urinary antiseptics. It is chemically stable and nontoxic and rarely does harm. Even when poorly tolerated it may cause only a slight strangury, microscopic hematuria or slight gastric irritation. Methenamine is excreted unchanged by the kidneys, becoming therapeutically active by the liberation of formaldehyde which takes place in the presence of an acid urine in the renal pelvis. Helmholz considers methenamine bactericidal in 24 hours to the *Bacillus coli* in an acid urine with a *p*H of 5. Methenamine will act efficiently only in the presence of acid urine. It is usually necessary to accompany the administration of the drug by acidifying agents such as sodium acid phosphate or ammonium chloride. Due to the action of urea-splitting bacteria which are often present in a mixed infection, a *p*H of 5 of the urine may be difficult or even impossible to maintain by the oral medication of these acidifying agents. It is possible to fortify the action of these acidifying agents by employing a high acid-ash diet. By maintaining a combination of these two measures the acidity of the urine may be assured.

Dosage: Methenamine, 0.3 to 0.6 Gm. (5 to 10 grains) four times a day, 1.3 to 2.6 Gm. (20 to 40 grains daily) should be administered in water or carbonated water after meals. Simultaneously, ammonium chloride, 0.3 to 1.3 Gm. (5 to 20 grains) or sodium biphosphate, 0.6 to 1.3 Gm. (10 to 20 grains), should be given.

Hexylresorcinol (Caprokal) was once heralded as a perfect urinary antiseptic. Its popularity has waned and at present it is seldom used. It was particularly recommended for infections of staphylococcic origin but possessed little effect upon the *Bacillus coli*.

Acriflavine: Acriflavine has given excellent results in the treatment of infected wounds and when used within the bladder. Its use has not been highly successful in the treatment of renal infections, not only from its limited bactericidal effect but also for its production of irritating gastrointestinal symptoms.

Pyridium, serenium, mallophene, ambizen, and other azo dyes have been synthetized as urinary antiseptics As a class their bactericidal properties have been proven to be very limited At present they are little used

Nitrohydrochloric acid therapy for the acidification of the urine in treatment of urinary infections was advanced in 1935 by Crance and Maloney It was proposed as a substitute for the ketogenic diet and is said to reduce the pH of the urine to 5 or less in less time than the ketogenic diet It is effective in combating infections due to the escherichia type of *Bacillus coli* and may be administered without hospitalization or without change of the ordinary diet The proponents state that it is difficult to sterilize the urinary tract infected with the aerobacter type of *B coli* but by the use of such therapy the pyuria and clinical symptoms disappear There are no apparent contraindications to the use of nitrohydrochloric acid It is well tolerated by patients during its administration and tests have failed to reveal any renal damage or intolerance A pH of 5 1 is necessary for efficient results The pH of the urine should be repeatedly checked during administration Its use should be continued for at least a week after the cultures become sterile

℞	Nitrohydrochloric acid (not the dilute)	18 cc. (ʒIVSS)
	Aquae dest q s ad	120 cc (℥IV)
Sig	One teaspoonful 4 cc (1 dram) in ⅔ of a glass of water followed by a full glass of water after meals and late at night	

Tests for acidity Estimation of pH may be simply and accurately done by the patient A daily written record may be kept by which a graph of the pH may be drawn

1 Nitrazine indicator (E R Squibb & Co) Test papers and comparative chart showing pH values by color intensities

2 Methyl red test (Coleman & Bell) Methyl red paper Red—pH below 5 5 yellow—pH above 5 5

3. Herrold's method of diacetic acid determination: One drop of 0.04 per cent of chlorophenol is added to 20 drops freshly voided urine. At *p*H 5.4 the color of the urine is not changed. This is the right degree of acidity to permit ketone bodies to exert their bactericidal power. A pink or red after addition of chlorophenol indicates a *p*H of above 5.4 and the urine is insufficiently acid.

4. Ferric-chloride test for diacetic acid. To 10 cc. of freshly voided urine an equal amount of ten per cent aqueous solution of ferric chloride is added. In the presence of diacetic acid a Bordeaux-red color develops.

Mandelic acid and its salts: In 1931 Clark made his first observations on the use of the ketogenic diet in the treatment of renal infections caused by the *Bacillus coli.* Fuller, in 1933, demonstrated that beta-oxybutyric acid was the principle bacteristatic agent in the urine of patients being treated by the ketogenic diet. Rosenheim, in 1935, introduced mandelic acid as a therapeutic agent, replacing beta-oxybutyric acid. It was demonstrated that mandelic acid, although acting similarly to beta-oxybutyric acid in its bacteristatic effect, was yet unoxidized by metabolism and was excreted unaltered in the urine. By this process of elimination and evolution it was possible to produce a nontoxic acid. This acid is bacteristatic in its action, is capable of resisting oxidization. It performs a therapeutic act similar to the unwieldy and unpleasant ketogenic diet. A concentration of mandelic acid of 0.25 to 1.0 and a urinary *p*H of 5.0 to 5.5 usually controls bacterial activity. Anything that hinders achievement of this concentration renders the treatment ineffective. The more acid the urine the greater the bactericidal effect of mandelic acid. In infections produced by the urea-splitting group of bacteria the alkalinity of the urine is such that the urinary *p*H seldom approaches 5.5 in

spite of the use of additional urine acidifiers In such instances the combined use of a modified ketogenic diet and mandelic acid may produce a sufficiently low *p*H of the urine to intensify the action of mandelic acid

Mandelic acid is generally used as the ammonium salt known as ammonium mandelate Originally Rosenheim used sodium mandelate supplemented by ammonium chloride to insure the proper *p*H of the urine The usual dose of ammonium mandelate is 3 Gm (45 grains) four times a day administered after each meal and at bedtime Fluids should be restricted to 1000 cc (one quart) a day and should be so rationed as to permit a constant urinary concentration of mandelic acid When employed in conjunction with the ketogenic diet the dosage of mandelic acid must be reduced Ammonium mandelate is given for a period of from five to ten days If by that time the urine is not free of bacteria a complete rest of 10 to 14 days from the drug should be permitted before giving a second course of treatment Long-continued administration of mandelic acid should never be permitted

Mandelic acid therapy may be attended by gastrointestinal symptoms such as nausea vomiting or diarrhea In the presence of known kidney or liver impairment mandelic acid therapy should be used with caution or not at all

Chemotherapy The list of bactericidal drugs used in the treatment of urinary infections has been greatly enhanced as the result of the introduction within recent years of a series of new chemotherapeutic agents such as sulfanilamide and its derivatives In 1935 Domagk produced prontosil (4 sulfonamide 0 2 4-diaminoazobenzol) by linking an azo dye with a sulfonamide group This combination was found to protect mice against streptococci The combination (prontosil) was one of many synthetized chemicals near the same chemical composition upon which synthesis

had or has now been done. Synthesis was continued until sulfanilamide was developed, a chemical agent which could be taken orally and which had definite bacteristatic qualities. Helmholz, in 1937, found that the urine obtained following administration orally of sulfanilamide was of sufficient concentration to be bactericidal to the *Staphylococcus aureus, Escherichia coli,* and the *Aerobacter aerogenes.* These findings, together with those of other investigators, stimulated the synthesis of further derivatives of the toxic sulfanilamide until the group included sulfapyridine, sulfathiazole, sulfamethylthiazole, sulfadiazine. Sulfathiazole and sulfadiazine are at present considered the less toxic and the more efficient of this group of compounds.

Unfortunately the sulfa group of drugs is now used quite universally but not always too intelligently or discriminately in the treatment of renal infections. They are administered too frequently without regard for the underlying causative factors of renal infection and regardless of the causative organisms that are present. It is only after failure to cure the infection that the causative factors are ascertained by a complete and thorough urological examination. Sulfanilamide is not used as extensively as when introduced, less toxic derivatives usually being administered. Several rather distressing and disagreeable complications may accompany the use of sulfanilamide: *Headache,* which is lessened on assuming a recumbent position; *nausea* may be constant but may be relieved by equal dosage of sodium bicarbonate; *cyanosis,* although it is not a serious complication, is due to sulfhemoglobinemia and methenoglobinemia. The presence of cyanosis does not require discontinuance of sulfanilamide and may be relieved by the administration of methylene blue, 0.3 Gm. (5 grains) three times a day. The more toxic manifestations of elevated temperature, dermatitis, hemolytic anemia, or agranulocytosis call for prompt discontinuance of the drug.

Dosage: Sulfanilamide should not be administered over long periods of time. When once begun the blood and urine levels of concentration should be taken at frequent intervals; likewise, frequent blood counts should be done. If high blood levels are found the dosage of the drug should be lessened to maintain a relative level of 8 mg. per 100 cc. The blood level of concentration should be maintained over 5 and under 10 mg. per 100 cc. If alteration in the blood picture is disclosed the drug should be stopped immediately. In certain instances relatively large doses may be administered with impunity, but in other individuals small doses may cause definite toxic reactions. An initial dosage of 4 to 5 Gm. (60 to 80 grains) daily in divided doses for two days, reduced to a maintenance dose of 1.5 to 2.6 Gm. (20 to 40 grains) for a maximum period of ten days, is suggested. Sodium bicarbonate of equal dosage should be given.

Sulfathiazole: Sulfathiazole is at present very extensively employed. The drug is less toxic than sulfanilamide. The drug shows an inclination to crystallization within the kidney during administration. Anuria and ultimate uremia have been known to occur due to complete obstruction of the renal tubules by the crystals. The toxic manifestations of the drug are few. Nausea may be observed following administration. Vomiting is unusual. The blood level should be ascertained at frequent intervals. The drug should be discontinued if a blood level of 12 mg. per 100 cc. is found.

Dosage: Sulfathiazole may be administered in an initial dose of 2.7 Gm. (45 grains) followed by a dose of 1 Gm. (15 graios) three times a day. Equal amounts of sodium bicarbonate should be simultaneously administered.

Penicillin: Penicillin is a recent addition to the list of bactericidal agents. Experimental studies are not complete. It has shown

low toxicity and has proven very effective in some cases of coccal infections of the kidney.

Dosage: The suggested dosage is 15,000 to 20,000 Oxford units initially administered followed by continuous intravenous injection of a solution delivering 5000 to 10,000 units per hour. The total dose in a 24-hour period may be reduced by half when the temperature has returned to normal. The drug should be continued for at least seven days after the temperature is normal.

Time and continued clinical trial will establish the effectiveness of penicillin in the treatment of renal infections.

The ketogenic diet: The ketogenic diet was introduced in 1931. Beyond all question the ketonuria produced by this diet definitely inhibits the activity of the *Bacillus coli* in infections of the urinary tract. In spite of its efficiency such a diet has several serious drawbacks. It requires hospitalization and rigid supervision for its efficient action. It is poorly tolerated by many individuals and frequently produces distressing general and gastrointestinal symptoms. Its use is contraindicated in the presence of impaired renal function.

The ketogenic diet consists of the ingestion of large amounts of fat, limited amounts of carbohydrates, but a normal ratio of protein. Ketonuria results from the nonoxidization of fat induced by an insufficient amount of glucose. It may be seen that this inadequate diet is low in carbohydrates, minerals, and vitamins; high in fat content but normal only in proteins and calories. For these reasons the diet should never be long continued.

Fuller, in 1933, showed that the bacteristatic effect of such a diet was due to the presence of levorotatory beta hydroxybutyric acid. Helmholz and Osterberg have shown that ketonuria, with a concentration of 0.5 per cent of beta hydroxybutyric in the presence of a urinary *p*H of 5.5 or less, is efficient in combating infec-

tion in the urinary tract by *Escherichia coli*. Crance has suggested that once the *Bacillus coli* has been shown to be the offending organism, subcultures of the urine should be done to determine which type of the *Bacillus coli* is present. *Escherichia coli* is present in about 75 per cent of instances and responds well to the ketogenic diet. The aerobacter type of the *Bacillus coli* is found in relatively 25 per cent of renal infections and does not respond to the diet.

The ketogenic diet, as originally outlined by Clark, is rigid and is difficult to apply. It was later modified. The original outline and its modification of the diet, together with the original suggestions of application, are given *in toto:*

Types of cases in which the ketogenic diet is indicated:

I. Initial or recurrent acute pyelonephritis or cystitis.

II. Chronic urinary infections without demonstrable gross pathologic change. Occasionally characteristic symptoms are present when the urine is grossly clear and microscopically negative for pus, but with bacilli in the stained sediment.

III Chronic urinary infection associated with pathologic change demonstrable by roentgenography, urography, or cystoscopy. The morbidity may often be reduced by active treatment of the urinary infection previous to operative treatment of the underlying pathologic condition. In some operations requiring only short hospitalization it may be beneficial to operate while the patient has an acid-ketone urine, thereby minimizing exacerbations of existing infections and the chances of new infection gaining a foothold.

IV. Urinary infection following operation.
  1. Operations on the urinary tract such as removal of calculi or vesical tumors and punch operation.
  2. Laparotomy, especially pelvic operations.
  3. Postoperative retention with overdistention.

V. Urinary infection following instrumentation such as passing of catheters, sounds or cystoscopes.

VI. Urinary infection following gonorrhea.

VII. Urinary infection in the presence of inoperable neoplasms.

I. *Bacillary Infections:*

1. Ketogenic diet (it must be followed as strictly as a diabetic diet).
   (*a*) It is quantitative. Daily diet prescription varies with individual. As a rule carbohydrate 15 Gm.; protein 0.6 Gm. per kilogram of normal body weight or 0.3 Gm. per pound of normal body weight; fat to meet caloric requirements, 225 to 275 Gm.
   (*b*) Duration
      1. Ketosis should develop in three to five days.
      2. As a rule patients who have the least tolerance for the diet, as evidenced by nausea, anorexia, weakness and malaise, develop ketosis most rapidly and obtain the quickest and most satisfactory results.
      3. If ketosis is not attained in 12 days, further effort probably will be unsuccessful.
      4. Not advisable to continue diet longer than two or three weeks after development of ketosis, without intermission

5 Yeast should be given if continued over long periods to avoid vitamin deficiency

6 Treatment will usually be longer if the upper part of the tract is involved

(*c*) Drugs Give throughout the period of the diet to insure adequate acidification of the urine

1 Solution of ammonium chloride is most effective

℞ Ammonium chloride 48 Gm (12 drams) water q s ad 500 cc. (16 ounces)

Sig 16 cc (4 drams) of the solution four times a day

2 In occasional cases a similar solution of ammonium nitrate may cause less discomfort to the gastrointestinal tract

3 If neither of the above solutions is tolerated ammonium nitrate 6 Gm daily 1 Gm every two hours the drug is put up in enteric coated tablets of 0 5 Gm each

(*d*) Estimations of patient s response

1 Test for diacetic acid Equal parts of ten per cent aqueous solution of ferric chloride and patient s urine should give a port wine color (Caution Aspirin will give a positive test)

2 Test for acidity Treatment is not effective unless the urine is acidified to a *p*H of 5 3 or less

3 Cultures of urine and stains of urine sediment every three or four days four or five negative cultures advisable

(*e*) Hypothesis as to mode of action.

Ketonurine is bactericidal or bacteristatic due to the B-oxybutyric acid

2. General measures:
   (*a*) Correction of constipation.
   (*b*) Removal of possible contributing foci of infection such as infected teeth or tonsils.
   (*c*) Exercise
   (*d*) No need either to push or to restrict fluids.

II. *Coccal Infections:* (These are frequently associated with bacilluria, particularly the *Streptococci fecalis* with bacilli of genus pseudomonas.)

1. Intravenous injection of 0.3 Gm. of neoarsphenamine every five days for six to eight doses (particularly for staphylococcic infections).
2. Bacteriophage (particularly for streptococcic infections).
3. Elimination of foci in:
   (*a*) Teeth or tonsils.
   (*b*) The prostate gland
4. Drugs.
   Methenamine with sufficient ammonium chloride or other acidifying agent to produce an acid urine (*p*H of 5.6 or lower).

Reasons for Unsatisfactory Results:

1. Poor renal function.
   (*a*) Hydronephrosis.
   (*b*) Pyelonephritis (chronic).
   (*c*) Polycystic kidney
2. Failure of ketonurine to develop.
   (*a*) Patient unable to digest fat.
   (*b*) Reason unknown.

3. Failure of low pH of urine to develop.
   (a) Reason unknown.
   (b) There are persons in apparently good general health and without demonstrable renal insufficiency whose kidneys do not put through: (1) ketone bodies in appreciable amounts, (2) urine of low pH even though CO_2 combining power of the blood is greatly reduced (30 volumes per cent).
4. Increased resistance of certain organisms to bactericidal agents.

   Aerobacter aerogenes is 10 to 20 times as resistant as *Escherichia coli.*
5. Failure of patient to cooperate in following the diet.
6. Insufficient period of treatment to get negative cultures (less than ten days of sufficient acidity and ketonurine) although symptoms markedly improved.

QUANTITATIVE DIET ORDER IS GIVEN IN TABLE

Carbohydrate, 15; protein, 52; fat, 225, calories 2300

Food	*Breakfast Grams*	*Dinner Grams*	*Supper Grams*	*Total Grams*	*Carbo hydrate*	*Grams Protein*	*Fat*
Vegetables (5% carbohydrate)	50	50	50	150	4.5	1.5	
Bran soya muffins	1	1	1	3	3.0	9	27
Butter	15	35	30	80			68
Cream (40% fat)	50	100	100	250	7.5	5	100
Bacon	20			20		5	10
Eggs	1			1		6	6
Meat		51	50	101		26	15.6
Total grams					15.0	52	226.6

SAMPLE MENUS:

Breakfast:	
Tomato juice	50 grams
Bran soya muffin	1
Butter	15 grams
Cream (40% fat)	50 grams
Bacon	20 grams
Egg	1
Dinner:	
Roast chicken	50 grams
Asparagus	50 grams
Bran soya muffin	1
Butter	35 grams
Cream (40% fat)	100 grams
Supper:	
Cold roast beef	50 grams
Boiled cabbage	50 grams
Bran soya muffin	1
Butter	30 grams
Cream (40% fat)	100 grams

A more simplified diet has been suggested by Clark and Keltz. The diet consists of combinations of the following foods:

Extra thick cream (40% fat)	Lettuce
Mayonnaise	Tomatoes
American cheese	Asparagus
Butter	Celery
Eggs	Salt and pepper
Bacon	

Group A: Egg Dishes

1. Eggnog

Egg	1
Cream (40% fat)	6 tablespoons
Water	5 tablespoons
Nutmeg	

Group B: Salads

1. Lettuce Salad

Lettuce	½ head
Mayonnaise	4 tablespoons

Group A Egg Dishes

2 SCRAMBLED EGGS

Eggs	2
Cream (40% fat)	7 tablespoons
Butter	3 teaspoons

3 EGG OMELET

Eggs	2
Cream (40% fat)	6 tablespoons
Butter	3 teaspoons

4 EGG CUSTARD

Eggs	2
Cream (40% fat)	6 tablespoons
Butter	3 teaspoons
Vanilla	2 drops

Group B Salads

2 LETTUCE AND TOMATO

Lettuce	1/8 head
Tomato	1 small
Hard boiled egg	1 yolk
Mayonnaise	5 tablespoons

3 ASPARAGUS SALAD

Asparagus	6 stalks
Lettuce	few leaves
Mayonnaise	4 tablespoons

4 COMBINATION SALAD

Lettuce	few leaves
Celery hearts	2
American cheese (grated)	2 tablespoons
Mayonnaise	4 tablespoons

Group C Cream Desserts

1 BAVARIAN CREAM

Gelatin	1 teaspoon
Cream (40% fat)	7 tablespoons

Whip the cream Soak gelatin in 1 teaspoon cold water then dissolve in 2 teaspoons hot water Add 2 drops of any flavoring When cooled add to the cream Place in mold and chill

2 GELATIN

Make plain gelatin as in 1 Use 7 tablespoons of unsweetened whipped cream over it.

3 7 tablespoons of cream—whipped with or without flavoring

Group D Beverages

Tea coffee or water with 4 tablespoons of cream If desired this amount of cream may be used with 2 cups of the beverage USE NO SUGAR OR MILK

DAILY MENU

BREAKFAST

1 One choice from Group A
2 One choice from Group D
3 8 slices of thin crisp bacon or 4 tablespoons of cream (40% fat)

DINNER AND SUPPER

1 One choice from Group B
2 One choice from Group A or C
3 One choice from Group D

In some cases it may be impossible for the patient to have especially prepared menus. Satisfactory results will be obtained if 1½ pints of 40 per cent cream and 6 eggs are prescribed as the daily food intake. The recipes and suggestions above in Groups A and D may be used.

CAUTIONS

1. Do not expect good results unless the diet is adhered to 100 per cent; 90 per cent adherence will not give 90 per cent results.
2. Do not allow any fluid other than the prescribed diet. Chewing tobacco, chewing gum, soda fountain drinks, fruits, milk of magnesia and sweet cathartics cause failure. Smoking is allowed.
3. Do not expect good results unless the *p*H of the urine remains less than 5.3.
4. Do not expect good results unless diacetic acid appears in the urine on daily tests; the greater the acidosis, the more effective the treatment.
5. Do not immediately discontinue the diet if patient feels nauseated or unable to take full diet. Often a day or two without food will relieve the symptoms, restoring the appetite and increasing ketosis without harmful effects. Patient burns his own fats instead of ingested fats.
6. Do not continue the diet too long without intermission (three weeks after ketosis develops). If symptoms of too severe acidosis or ketosis develop they can readily be relieved by giving alkalies and sugars.
7. Do not force fluids. Encourage normal intake of fluids.
8. Do not limit the patient's ordinary activities. Exercise increases the ketosis.
9. Do not forget, in mixed infections, to treat the coccal type also.

10 Do not be misled by severe prostatitis, assuming that it and the accompanying posterior urethritis account for all the symptoms There may be a bacillurin too Culture the urine

11 Do not forget treatment of the prostate gland if it is badly infected This prevents reinfection of the vesical urine

12 Do not forget lavage of the bladder although this is not imperative

13 Do not forget the teeth, tonsils and other foci

14 Do not forget measures to correct constipation Mineral oil and agar are often indicated

15 Do not forget the possibility of other pathologic changes previously overlooked If there is no improvement of symptoms after two weeks of sufficient acidity and ketonurine, advise cystoscopy, intravenous urogram, inoculation of guinea pigs or other indicated diagnostic procedures

16 Do not forget to examine microscopically and to culture the urine in all cases (*a*) in which the second glass is cloudy, and (*b*) in which there are characteristic symptoms even though the second glass is clear

High Acid Ash Diet The high acid ash diet is used in instances where it is deemed advisable to change and maintain an acid urine Such a diet should fulfill the normal food requirements but an excess of acid ash should be produced

A diet meeting those requirements has been outlined by Higgins and is presented verbatim

High Vitamin Acid Ash Diet The purpose of this diet is to furnish an adequate high vitamin diet in which the total acid ash exceeds the total basic ash To accomplish this it is absolutely necessary that no salt be used for seasoning foods either in cooking or at the table The following foods, in the amounts designated must be included in the diet daily

A. Acid-Ash Foods (minimum amounts).

I. Cereal in one of the following measured servings (2 cc. excess acid-ash).

Cornflakes	1 cup heaping
Cornmeal, cooked	⅔ cup
Farina, cooked	⅔ cup
Oatmeal, cooked	½ cup
Puffed wheat	1 cup scant
Puffed rice	1 cup heaping
Rice, cooked	½ cup scant
Shredded wheat	½ biscuit

II. Meats. Any two of the following measured servings (12 cc. each).

Beef, loin, medium fat	4" x 4¼" x ½"
Chicken, broiled	½
Chicken, stewed	breast or thigh, plus leg
Cheese, cheddar	3½" x 2" x 1"
Codfish, fresh, cooked	¼ cup
Eggs	2
Frankfurters, large	2
Halibut	4" x 2" x 1"
Ham, fresh	4½" x 3" x ¼"
Heart, beef	2½" x 3" x 1"
Kidney, veal	¾ cup
Lamb chop	3 medium size
Lamb roast	5" x 5" x ¼"
Liver, beef	3" x 6½" x ½"
Mackerel, fresh	2" x 4" x 1"
Oysters, very large	3
Pork chop, thick	1
Salmon, fresh	3" x 4" x ¾"
Salmon, canned	½ cup packed
Trout	2½" x 3" x 1"
Turkey, two slices	2" x 3" x ¼"
Veal chop	1
Veal roast	3" x 2½" x ⅛"
White fish	2¼" x 3" x 1"

III. Bread—whole wheat, five slices (2 cc. each).

IV. Eggs—two (5 5 cc. each).

V. Miscellaneous—any one of the following measured servings (2 cc.).

Macaroni	¾ cup
Spaghetti	½ cup
Rice	½ cup
Corn	½ cup
Plain cake	1¾" x 1¾" x 1¼"

B. Alkaline Ash Foods (maximum amounts).

I. Milk—one pint (7.2 cc.).

II. Cream—¼ cup (0.3 cc.).

III. Fruits and vegetables (see below). (Not to exceed 25 cc.)

C. Concentrated Vitamin Foods.

I Yeast—two cakes.

II Cod liver oil—two tablespoons, or haliver oil, two capsules before each meal.

III. Wheat germ—two tablespoons to be added to cereal.

Fruits and vegetables shall be chosen from the following lists only. Any combination of fruits and vegetables may be selected, but the total excess basic-ash in the selected combination must not exceed 25 cc. daily

Fruit	*Amount*	*cc. of excess basic-ash*
Watermelon	2½" x 2½" x ½"	2.7
Grapes	½ cup or 24 grapes	2.7
Pear	1 medium	3 6
Apple	1 small	3 7

Fruit	*Amount*	*cc. of excess basic-ash*
Grape juice	½ cup	3.9
Lemon juice	½ cup	4.1
Cherry juice	½ cup	4.4
Orange juice	½ cup	4.5
Raspberry juice	½ cup	4.9
Peach	1 medium	5.0
Lemon	1 medium	5.5
Banana	¾ cup or ½ large	5.6
Orange	1 medium	5.6
Cherries	⅔ cup	6.1
Apricots	2 medium	6.8
Pineapple	⅔ cup diced	6.8
Muskmelon	½ cup	7.5
Rhubarb	½ cup	8.6

Vegetable	*Amount*	*cc. of excess basic-ash*
Asparagus	½ cup	0.8
Green peas	¾ cup	1.3
Onions	½ cup	1.5
Pumpkin	½ cup cooked	1.5
Turnips	½ cup cooked	2.7
Squash	½ cup mashed	2.8
Radishes	10	2.9
Mushrooms	½ cup canned	4.0
Cauliflower	⅔ cup cooked	5.3
String beans	⅔ cup cooked	5.4
Tomatoes	½ cup	5.6
Cabbage	⅔ cup cooked, 1½ raw	6.0
Tomato juice	½ cup	6.2
Sweet potato	½ medium size	6.7
White potato	1 potato 2½" diameter	7.0
Lettuce	¼ head or 16 leaves	7.1
Celery	4 stalks or ¾ cup	7.8
Cucumber	⅓ cup sliced	7.9
Rutabagas	½ cup mashed	8.5
Carrots	⅝ cup	10.8
Beets	⅔ cup	10.9

The following list contains a few striking examples of foods which *must be omitted* because of their extremely high basic ash content

Almonds, beet greens, dandelion greens, figs, molasses, olives, parsnips, raisins, spinach dried fruits and vegetables

HIGGINS HIGH VITAMIN ACID ASH MENU

BREAKFAST

Fruit	Grapes
Cereal and wheat germ	Oatmeal and 2 tablespoons wheat germ
Eggs	2 scrambled
Bread whole wheat toasted	2 slices
Salt free butter	2 squares
Milk	½ pint
Cream	¼ cup
Sugar	As desired
Beverage	Coffee

LUNCH

Meat	Veal chop
Rice or substitute (see misc.)	Steamed rice
Vegetable or salad	Sliced tomatoes
Fruit	Baked apple
Bread whole wheat	1½ slices
Salt free butter	2 squares
Beverage	Milk, 1 glass

DINNER

Meat	Roast beef
Vegetable	Potato
Vegetable	String beans
Dessert	Tapioca cream pudding

Bread, whole wheat	1½ slices
Salt-free butter	2 squares
Cream	As desired
Sugar	As desired
Beverage	As desired

Note: One pint of milk is to be used each day in any form. In addition, the following acid and neutral foods may be used as desired. Acid fruits, cranberries, flour (plain cookies), pastry with custard or allowed amounts of fruit fillings, English walnuts, popcorn (no salt), unsalted peanuts, unsalted crackers.

Neutral foods: Sweet butter, candy (no chocolate bars), lard, olive oil, salad oil, cornstarch, mayonnaise, sugar, tapioca, tea, coffee, or Kaffee Hag, Postum.

High Alkaline-Ash Diet: In the treatment of certain urinary infections, particularly those of the lower urinary tract with its accompanying symptoms of frequency and urgency of urination, considerable relief of the irritating symptoms may be often gained by the production of an alkaline urinary reaction without the use of medicaments. Such a diet should be of normal food requirements but one producing an excess of alkaline-ash.

HIGGINS HIGH VITAMIN ALKALINE-ASH DIET:

A. Alkaline-Ash Foods:

I. Milk—one quart.

II. Cream—½ cup.

III. Fruits and vegetables—any combination of fruits and vegetables may be selected from the following list but the excess basic-ash must total at least 38 cc. daily.

Vegetable	*Amount*	*cc. of excess basic-ash*
Celery	4 stalks or ¾ cup	7.8
Cucumber	⅓ cup	7.9
Rutabagas	½ cup	8.5
Carrots	⅝ cup	10.8
Parsnips	½ cup	11.9
Beans, lima, green	½ cup	14.0
Chard	½ cup	15.7
Beans, navy, dried	¾ cup	26.1
Spinach	½ cup	27.0
Beet greens	½ cup	27.0
Beans, lima, dried	⅔ cup	41.6

B. Acid-Ash Foods:

I. Meats. Any two of the following measured servings.

Beef, loin, medium, fat	4" x 4¼" x ½"
Chicken, broiler	½
Chicken, stewed	breast or thigh, plus leg
Cheese, cheddar	3½" x 2" x 1"
Codfish, fresh, cooked	¼ cup
Eggs	2
Frankfurters, large	2
Halibut	4" x 2" x 1"
Lamb roast	5" x 5" x ¼"
Ham, fresh	4½" x 3" x ¼"
Heart, beef	2½" x 3" x 1"
Kidney, veal	¾ cup
Lamb chop	3 medium size
Liver, beef	3" x 6½" x ½"
Mackerel, fresh	2" x 4" x 1"
Oysters, very large	3
Pork chop, thick	1
Salmon, fresh	3" x 4" x ¾"
Salmon, canned	½ cup packed
Trout	2½" x 3" x 1"
Turkey, two slices	2" x 3" x ¼"
Veal chop	1
Veal roast	3" x 2½" x ⅛"
White fish	2¼" x 3" x 1"

II Eggs—one

III Bread—whole wheat two slices

IV Cereal—any one of the following measured servings

Cornflakes	1 cup heaping
Cornmeal cooked	2/3 cup
Farina cooked	2/3 cup
Oatmeal cooked	1/3 cup
Puffed wheat	1 cup scant
Puffed rice	1 cup heaping
Rice cooked	1/3 cup scant
Shredded wheat	1/2 biscuit

C Concentrated Vitamin Foods

I Yeast—two cakes

II Cod liver oil—two tablespoons or haliver oil, two capsules before each meal

In addition, the following alkaline and neutral foods may be used as desired

Alkaline foods Dairy products including all cheese * soups except when made from meat stock almonds molasses, olives

Neutral foods Sweet butter, candy (no chocolate) cornstarch lard, salad oil sugar coffee, tea Kaffee Hag Postum, olive oil mayonnaise tapioca

The following list contains a few striking examples of foods which *must be omitted* because of their extremely high acid ash content

Meat broths all breads and crackers except as listed above, all pastries and rich desserts, cranberries peanuts walnuts pop corn flour

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* Usually regarded as acid.

HIGGINS HIGH VITAMIN ALKALINE MENU

Breakfast:	
Fruit	Orange juice—½ cup
Cereal	Farina—⅔ cup, cooked
Egg	1
Whole wheat toast	1 slice
Butter	As desired
Cream	½ cup
Milk	1 glass
Lunch:	
Meat	Cold sliced lamb, one serving
Potato	Baked, one serving
Vegetable	Celery, one serving
Bread, whole wheat	½ slice
Butter	As desired
Milk	1 glass
Fruit	Canned peaches—1 serving
Dinner:	
Meat	Roast beef—one serving
Potato	Sweet potato—one serving
Vegetable	Cauliflower—one serving
Bread, whole wheat	½ slice
Butter	As desired
Milk	1 glass
Dessert	Vanilla ice cream
8:00 P.M.	
Milk	1 glass

C. *Vaccines and Bacteriophage:* Considerable interest in the use of vaccines in the treatment of urinary infections has existed for several years but the results obtained have not been particularly satisfactory.

Cummings has prepared autogenous vaccines of bacteria to which a patient is hypersensitive and has shown such vaccines to

be extremely powerful, exhibiting a high degree of specificity and even minute doses occasionally produce violent systemic reactions Realizing that the reactions were so severe vaccines were prepared by repeated dilution until they contained only five to ten organisms for each strain of bacteria per cubic centimeter Even with such dilution, he has observed an occasional reaction to a dose as small as 0 05 cc

Bacteriophage therapy, although very limited, has been of some aid in the treatment of urinary infections Wehrbein and Nerb have reported seven cures in ten cases treated for acute pyelonephritis, but only 5 of 24 cases treated for chronic infection Wehrbein states that the bacteriophage, to be effective, must be brought into massive direct contact with the offending organism The bacteriophage should be placed in as large a quantity and in as concentrated a form as possible in the infected area The renal pelves should be filled with the phage solution He further states that if the first application is not successful further applications of such treatments are useless

D *Indwelling Ureteral Catheter* The use of an indwelling ureteral catheter, with ureteral dilatation and pelvic lavage, is indicated in subacute and chronic renal infections The indwelling catheter is particularly useful in the maintenance of normal and adequate drainage in pyelonephritis of pregnancy or in certain types of ureteral obstruction It is imperative, in the use of this treatment to maintain constant drainage by frequent irrigation with small amounts of sterile water or saline solution If the catheter is permitted to become blocked, the resultant dilatation of the renal pelvis not only is painful but adds materially to the continuance of the infection Undoubtedly much benefit is derived from ureteral catheterization by mechanical dilatation of the ureters which would tend to relieve any congestion or cellular

edema present. Lavage of the renal pelves is indicated in subacute or chronic infections but should never be used in the acute stage of such infections. Solutions of silver nitrate, 1:1000 to 1:5000 (the weaker solutions are to be preferred); acriflavine, 1:200 to 1:400; mercurochrome, one per cent, may be used advantageously.

E. *Surgery:* Surgery is indicated in certain instances of renal infection depending upon the individual findings of each case. The presence of a congenital anomaly or the presence of removable obstructive factors; virulence and nature of the infection; the condition and functioning ability of the opposite kidney are guiding factors in the management of each individual case. All the factors must be taken into consideration and analyzed before surgical procedures may be instituted.

FOCAL PYOGENIC INFECTIONS OF THE RENAL CORTEX—*CORTICAL ABSCESSES—CARBUNCLE*

Focal renal infections are so grouped or classified as a clinical entity induced by a distant focus of infection arising from hematogenous origin. Usually these focal infections are the result of staphylococcic infection. The streptococcus may occasionally be found as the causative organism. Ascending urinary infections by the *Bacillus coli* may produce abscesses within the kidney. Such abscesses are seldom primarily observed on the renal cortex as the infection usually spreads from the renal pelves along the line of *the blood vessels or by backflow ultimately to reach the cortex* secondarily. Ascending infections present the definite urinary findings of great numbers of leukocytes and some red cells. In blood-borne or hematogenous infections of the kidney, the bacteria enter the small terminal blood vessels and are carried directly to the cortex with the formation of localized metastatic lesions on the surface of the kidney.

It cannot be explained why the *Staphylococcus pyogenes aureus* is the usual causative organism Mixed infections do occur The *Staphylococcus albus* and *Streptococcus pyogenes* or *hemolyticus* may be identified, but the *Staphylococcus pyogenes aureus* is the most frequently offending organism producing cortical infections

The infection of the kidney follows, or is associated with a blood stream infection which may vary from a mild bacteremia to a severe septicemia Secondary to this generalized blood stream infection, regardless of its severity, abscesses of various parts of the body may occur Among children the bones are elective sites of metastatic bacterial invasion as well as the kidneys In adults it is usually the soft tissues only that are involved, the kidney in particular

The site of the original infection is usually a peripheral or superficial suppuration, such as a boil, furuncle, felon, or superficial infected wound From the primary focus in the skin the stapylococcus is carried by the blood stream to the kidney cortex Depending upon the virulence of the organism and resistance of the individual to this particular organism, several degrees of intensity of renal cortical infection may be observed Each is a stage of infection of the renal parenchyma and cortex, all tending to the same ultimate ending destruction of the renal parenchyma and usually the ultimate, perinephritis

(*a*) Focal diffuse staphylococcic nephritis (Without localization or abscess formation) The kidney may present a wide degree of inflammatory reaction varying from a very mild hyperemia to an acute fulminating type of inflammation In the mild form, the lesion is that of varying degrees of inflammation without suppuration In the fulminating form, the kidney shows an intense, diffuse inflammation It is swollen, soft and discolored In the mild type, spontaneous recovery is usual In the fulminating type, immediate nephrectomy is the only life saving measure to be considered

(*b*) Multiple staphylococcic cortical abscesses: These abscesses are small and as the name implies, are multiple. The abscesses may be scattered over the entire kidney surface or may be localized in one particular area. The kidney presents a spotted appearance, the slightly hyperemic surface studded by small, yellowish, multiple abscesses (PLATE LXVIII). The symptomatology produced by such a condition in the early stages may be slight. There may be a slight tenderness at the costovertebral angle and a mild, irregular, febrile course. The urine may show a few red cells, an occasional leukocyte and a slight albuminuria. There is a tendency to renal dysfunction, limited to the affected kidney. The condi-

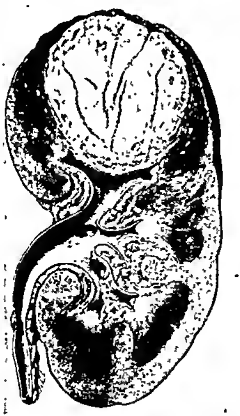


Fig. 315—Carbuncle filling upper pole of left kidney.
(Courtesy of Dr. Vincent J. O'Conor and *Journal of Urology*, 1933, 30 1.)

tion usually progresses to perirenal suppuration and abscess formation but may gradually subside.

(*c*) Carbuncle of the kidney (circumscribed metastatic suppurative nephritis) Carbuncle of the kidney is a circumscribed multilocular abscess of the renal parenchyma (FIG. 315). The abscess is metastatic, usually arising from a primary yet peripheral site of infection. The *Staphylococcus pyogenes aureus* is the usual causative organism Like other types of renal cortical infections the condition is progressive, usually leading to perirenal suppuration and abscess formation. Such a formation may rupture into a calyx with complete evacuation and ultimate healing. The condition usually runs a more protracted course, causing a train of vague symptoms. The kidney is subjected to a localized but definitely progressive suppuration which is limited externally by the fibrous

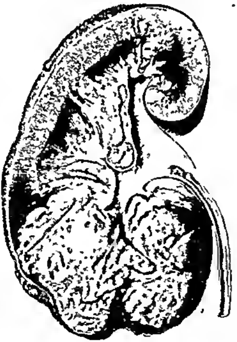


FIG 316—Carbuncle completely filling lower pole of left kidney (Courtesy of Dr Vincent J O Conor and *Journal of Urology* 1933, 30 1)

PLATE LXVIII—Multiple staphylococcic cortical abscesses. Actual color photograph showing the small abscess in the parenchyma of the kidney.

renal capsule Being progressive, the suppuration may involve a third or a half of the kidney before rupture of the fibrous capsule occurs (Fig 316) Following the rupture of the capsule, a perinephritis occurs with all its associated symptoms

Symptoms—There are no pathognomonic symptoms of renal carbuncle In fact, the symptomatology is usually so vague that diagnosis is not only difficult but occasionally impossible The symptoms may be of such slight intensity that a renal carbuncle may exist for months before its presence is discovered The actual onset is usually marked by pain in the general region of the flank Rarely is the pain severe at the onset but it is usually diffuse The acute onset has been mistaken for acute pulmonary or intra abdominal affections Malaise, remittent chills and fever, loss of weight, progressive anemia, or an associated pleuritic irritation are not uncommon (Fig 317) Symptoms referable to the kidney may only occur with extension of the suppurative process to the

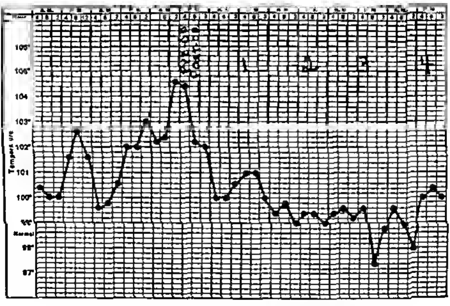


Fig 317—Temperature chart in the presence of renal carbuncle Temperature fell immediately following drainage

perirenal tissue. Graves and Parkins, in an analysis of 67 cases, state that . . . "The whole clinical picture, conspicuously vague, may closely simulate that of a blood stream infection. In fact, in our opinion, an underlying septicemia is probably often present. The obscurity of the lesion is well emphasized by the preliminary diagnosis suggested in the 67 cases under consideration. Some of these were: Typhoid fever, prostatic obstruction, hydronephrosis, appendicitis, pleurisy, pneumonia, nephroptosis, abdominal lymphadenitis, tumor, pyelonephritis, influenza, cholelithiasis, and septicemia."

As the condition progresses along its relatively chronic course, backache, tenderness in or near the region of the kidney, resistance to palpation in the overlying muscles, moderate, irregular fever, loss of appetite and weight, slight yet progressive anemia with an associated leukocytosis are suggestive symptoms of renal carbuncle. In contrast, localizing symptoms in the renal area may be marked while the suppurative process is limited to the cortex. This pain over the renal area is due to the tension within the fibrous renal capsule and subsides quickly in the event of rupture of the abscess into the perirenal tissues.

Diagnosis: The diagnosis of renal carbuncle may be so difficult as to be impossible, or the symptoms may be so vague as to be completely overlooked. Undoubtedly many instances of localized cortical abscess have undergone complete resolution with complete healing without ever having been recognized as such. On the other hand, many instances of cortical infection have progressed through successive stages of septicemia to a fatal termination, without ever presenting renal symptomatology.

Possibly the greatest single factor is the history of a peripheral infection (boil, carbuncle or felon) in the recent past. In 42 of 67 cases of renal carbuncle, reviewed by Graves and Parkins, a history of "superficial pyogenic infection" was elicited.

The clinical confusion in arriving at a definite diagnosis is due not only to the almost complete lack of urinary symptoms and urine findings, cystoscopy and ureteral catheterization, but also to the vague nature of the symptoms portrayed *Ureteral catheterization* will demonstrate the patulousness of the ureters, otherwise little information is to be gained *Cystoscopy* may reveal a diminution of function on the affected side as compared with the renal function on the opposite side The depression of renal function is considered by Graves and Parkins to be of the greatest importance in the differential diagnosis of this condition

Retrograde pyelography may reveal a distortion of the calices but such distortions are not typical or pathognomonic of the condition The filling defects observed may well be produced by other pathological processes such as cysts or neoplasms

Intravenous urography may be of some benefit, but in instances where renal function is lowered, proper excretion of the dye will not be of sufficient concentration to be well visualized

Possibly the greatest x ray evidence afforded is the limitation and fixation of the diaphragm on the affected side

Treatment The treatment of renal carbuncle is surgical The mode of treatment is governed by the findings in each individual instance Incision and drainage of the perinephritic abscess should be followed by nephrectomy, the enucleation of the carbuncle or primary nephrectomy

Diffuse Suppurative Nephritis

Diffuse suppurative nephritis is usually bilateral and is associated with a generalized septicemia The rapid, fatal termination of the clinical picture is such that generally the patient succumbs to the systemic infection before localizing kidney symptoms develop In some instances of acute unilateral, hematogenous renal infections of a hyperacute or fulminating nature, nephrectomy is

indicated. It is imperative to prove the opposite kidney to be uninfected. In instances of acute or subacute hematogenous renal infections, supportive treatment, blood transfusions, and the oral administration of such drugs as sulfathiazole or sulfadiazine may aid in combating the infection.

PERINEPHRIC ABSCESS

Perinephric abscess is a suppuration of the fibro-fatty tissue (perinephrium) between the kidney and the renal fascia (Gerota's fascia). This suppurative process may be located outside of the renal fascia. It is then considered a perirenal abscess. These terms are used to indicate the relation of this suppurative process to the capsule. Clinically, these lesions are indistinguishable.

As in all inflammations, there are wide variations in the extremes of tissue reaction to bacterial invasion. When the perirenal inflammation is extensive and localizes quickly, suppuration occurs with the development of abscess formation. If the infection is chronic, as is seen in advanced but protracted renal lesions (pyonephrosis, tuberculosis) the perirenal infection does not quickly progress to suppuration but undergoes fibrosis. Dense adhesions to the kidney and to the surrounding structures are evident. Both conditions result from inflammation of the perirenal tissue, but the intensity of the bacterial invasion varies in either instance.

Perinephric abscess, *per se,* is a suppuration of perirenal tissue induced by the implantation of bacteria. The usual causative organism is the *Staphylococcus pyogenes aureus,* although *Staphylococcus albus,* streptococcus, pneumococcus, and various other organisms are occasionally isolated. The route of the invading organism to the perirenal tissues is subject to rather wide and diversified opinion. Vermooten believes that there is present a separate arterial supply from the renal artery to the perinephrium,

which would permit bacteria to be directly implanted in the peri renal tissue Most observers believe that there is a direct implanta tion of the organisms from the renal cortex into the perinephrium Regardless of the mode of invasion the clinical picture presented is the same

Symptoms and Diagnosis Perinephric abscess is usually unilat eral Cases have been reported where the condition was bilateral The condition is more frequently observed in men than in women probably due to the fact that men seem to be more frequently subject to peripheral skin infections than are women

There are no pathognomonic symptoms of perinephric abscess For this reason the diagnosis is usually not made until the condi tion is rather far advanced In the early stages even in the event of an acute onset there are few suggestive symptoms Fever leuko cytosis and pain in the renal region are commonly present In the event of fulminating onset chills fever leukocytosis nausea and vomiting and abdominal disturbances may be observed Exami nation discloses marked tenderness in the loin muscular rigidity and eventually a palpable mass in the loin The onset may be so gradual that the only manifestations are those of general but pro gressive weakness and unexplained and irregular fever There is usually definite tenderness at the costovertebral angle and muscu lar rigidity As the condition progresses flexion of the thigh may be so extreme as to prevent extension

Perinephric abscess may be mistaken for psoas abscess and if long continued may find its way to rather distant points Peri nephric abscess may rupture and drain through the skin at the renal area into the inguinal region or may perforate the dia phragm causing an empyema or even a bronchial fistula A reno colic fistula may be formed by the rupture of a perinephric abscess into the intestines

Examination of the urine may reveal little or nothing of im

portance as the tubules and collecting portion of the kidney are not usually involved in the inflammatory process. Occasionally, staphylococci may be isolated from the urine coming from the kidney of the affected side.

Cystoscopy is of little value. Possibly the greatest information afforded is from the individual renal function tests. These tests may reveal some slight dysfunction on the affected side. This is true only when cortical abscesses are present, but it is not true when the cortical lesions have healed and only the perinephric abscess is present.

Fig. 318—Roentgenogram. Perinephritic abscess. Note absence of right psoas shadow, a characteristic finding of perinephritic abscess formation.
(Temple University Hospital, Acc. No. 25890.)

The roentgenogram is one of the most important factors in making a diagnosis of perinephric abscess (Fig 318) The findings of the roentgen ray may be long in evidence before the clinical picture is such as to make an accurate diagnosis by physical examination X rays of the abdomen including the renal areas usually reveal four salient diagnostic points (1) Obliteration of psoas shadow (2) effacement of the renal shadow (3) curvature of the lumbar spine away from the affected side (4) fixation of the diaphragm All are pertinent diagnostic signs in the presence of perinephric abscess Mathe has called attention to the loss of mobility of the kidney which may be demonstrated by the contrast pyelograms made in the Trendelenburg and in the upright positions

Treatment The only treatment for perinephric abscess is incision and drainage Secondary nephrectomy may be indicated in the presence of destructive lesions of the kidney

REPLACEMENT LIPOMATOSIS OF THE KIDNEY

Replacement lipomatosis of the kidney is an invasion and replacement of the functioning parenchyma by fat The condition has been somewhat confused with lipoma of the kidney Lipomata or fatty tumors are usually small yellow encapsulated nodules in the cortical layer just beneath the fibrous capsule of the kidney Replacement lipomatosis possesses none of these characteristics but is a definite infiltration of fat which invades the renal tissues Replacement lipomatosis begins at the hilus of the kidney and extends toward the periphery replacing the destroyed or atrophied parenchyma by fatty tissue (Plate LXIX)

The condition was first described in 1778 by Barder At autopsy he observed a pyelonephritis with an associated calculus and a fatty infiltration of the kidney

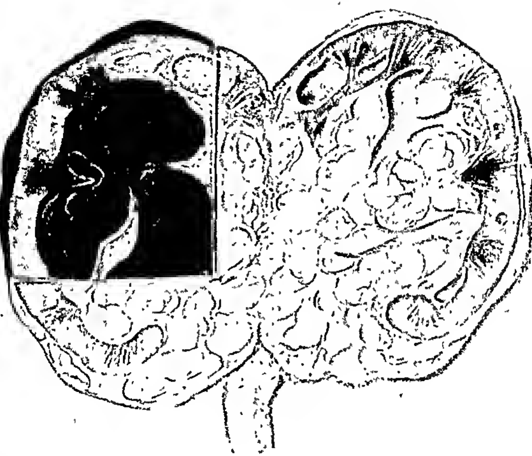


PLATE LXIX—Replacement lipomatosis of the kidney. Extensive deposits of fatty tissue have replaced the major portion of the kidney tissue. The upper left-hand portion of the kidney has been stained with Suden III.

Kutzman, in 1931, in an analysis of 33 cases, found evidences of infection with associated calculous formation in 79 per cent. He concluded that . . . "the process is probably one of invasion by the perirenal, peripelvic and hilus fat along the great vessels through the renal hilus, following a destruction of the renal parenchyma by calculus and infection." However, Young reported a case in which there was neither infection nor calculus present, although . . . "there was slight evidence of perinephritis."

Symptoms and Diagnosis: There are no pathognomonic signs or symptoms of replacement lipomatosis. The condition, when present, presents only one definite finding; namely, a functionless kidney. Pain may or may not be present. If present, it is not typical in character.

Urinalysis reveals a normal urine because the unaffected kidney will be doing the work of both sides. The affected kidney, being functionless, the pelvis occluded by fat, there cannot be the usual finding of pyuria, common in infections of the kidney.

Cystoscopy: There are no evidences within the bladder of the existing condition. The presence of a functionless kidney on one side and a normally functioning kidney on the other will be ascertained by the intravenous administration of indigo-carmine.

Retrograde pyelography may not be of value because of the extreme atrophy of the kidney. The radiopaque medium cannot be injected into the renal pelvis. If injected, a bizarre pyelogram will be visualized, which does not present a typical or characteristic appearance.

Intravenous urography may likewise be worthless because of the presence of a functionless organ.

Treatment: Since the kidney affected by replacement lipomatosis is functionless and may show evidence of extreme infection and calculous formation, nephrectomy is the only treatment.

XXIII

MEDICAL DISEASES OF THE KIDNEY

There is no one subject in which there is so great confusion existing as in the so-called medical diseases of the kidney. This confusion is undoubtedly due to the utter impossibility of definite classification of the condition, or a true correlation of the various clinical types and the pathology, both gross and microscopic.

The underlying factors in the production of nephritis are not well understood, as the lesions produced may fall into one type or into two or three types existing simultaneously within the same diseased organ. These three pathological lesions observed in nephritis may be classified as: (1) Hemorrhagic or inflammatory; (2) degenerative or necrotic; (3) sclerotic.

Many of the so-called surgical diseases of the kidney may be complicated by the so-called medical diseases of the kidney, or a surgical condition of the kidney may exist on one side and an extreme medical condition exist on the opposite side. It is therefore imperative that the urologist be able to recognize and cope not only with the surgical conditions of the kidney, but with the medical conditions as well.

The structure of the kidney consists of three primary divisions or elements:

1. Vascular element, which consists of the renal arteries, veins and glomerular tufts.
2. The interstitial tissues.

3 The epithelial tissues which include Bowman s capsules and the membranous lining of the renal tubules

Each of these primary divisions may be affected individually, although it is usual that all are involved as the disease advances

GLOMERULONEPHRITIS

First Stage Acute Diffuse Glomerulonephritis

Acute diffuse glomerulonephritis is a bilateral inflammation primarily involving the glomeruli of the kidneys Secondary damage to the renal tubules and to the interstitial tissues occurs at a later stage of the disease As a result of the extreme degree to which the pathology and symptomatology vary glomerulonephritis has been erroneously described under three different forms (1) Acute nephritis (2) subacute nephritis (3) chronic parenchymatous nephritis chronic nephritis or chronic interstitial nephritis As a result of the work of Lohlein, Volhard and Fahr, Bell and others it has been shown that these three conditions are three separate advancing stages of the same pathological process Clinically the first stage is characterized by the presence of casts and red cells in the urine the signs of acute renal insufficiency, edema and varying degrees of hypertension The second stage is characterized by edema and albuminuria The third stage is characterized by hypertension and renal failure Should the kidneys be subjected to repeated infections they may pass through all three stages Pathologically Boyd considers the first stage to be one of proliferation the second stage one of degeneration and the third stage one of atrophy and scarring Recovery is usual after the first stage the prognosis poor in the second stage the third stage is terminal

Acute diffuse glomerulonephritis is bilateral and follows bacterial infection The infection is extrarenal and not within the kidney itself It is usually induced by staphylococci or streptococci

and follows many different types of extrarenal infections (tonsillitis; the exanthemata, principally scarlet fever). The kidney itself is not the site of an actual suppurative infection as is seen in pyelonephritis following a localized staphylococcic or streptococcic infection. In acute diffuse glomerulonephritis, the kidney is the site of an inflammation undoubtedly produced by diffusible toxins rather than the direct implantation of bacteria into the kidney tubules.

There is usually a latent period of from one to three weeks between the initial infection and the apparent onset of the kidney involvement. The onset is marked by puffiness about the eyes; scanty amounts of bloody urine; loss of appetite; nausea, perhaps vomiting; dull pain in the lumbar region; slight fever and pallor. Hypertension from a mild to a moderate degree may be present. A moderate degree of retention of nonprotein nitrogen may exist. Edema of the face, hands, feet, or genitalia may be seen. The diagnostic triad of hematuria, edema, and hypertension, characteristic of acute glomerulonephritis, is usually present. Although all the classical triad of symptoms may not be present, the picture presented is usually dominated by one of them. When hematuria is the most predominant symptom, the presence of calculus, neoplasm or tuberculosis of the kidney may be suspected.

Pathology: The kidney is usually of normal size, but it may be enlarged. The capsule of the kidney is of normal thickness, is *tense and strips readily. The cortical surface of the kidney is pale.* When sectioned, the renal cortex presents a pale, swollen appearance. The glomeruli appear as bright red dots or points. The pyramids of the kidney are deeply congested. Microscopically, the glomeruli show the essential lesions, although the tubules may secondarily show definite changes. All the glomeruli are involved. There are endothelial proliferation and hyalin formation which

occlude the loop of Henle with a resulting glomerular ischemia (Fig 319)

Symptoms and Diagnosis: The urologist is often called upon to aid in making a differential diagnosis

Cystoscopy There is nothing pathognomonic in the cystoscopic

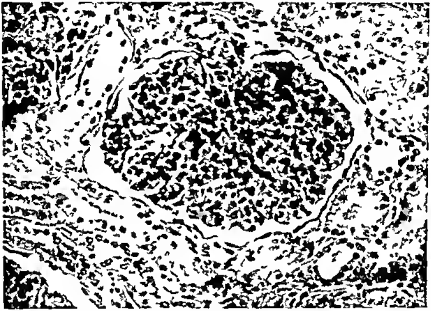


Fig 319—Acute glomerulonephritis Photomicrograph showing early changes The capillaries of the tuft are avascular but the number of cells increased as a result of endothelial and epithelial proliferation Note early diffuse polynuclear leukocytic exudation
(Courtesy of Dr Lawrence W Smith and Dr Edwin S Gault)

appearance of the bladder in glomerulonephritis Considerable important data may be gained by cystoscopy and ureteral catheterization with the collection of individual specimens for analysis and study Occlusion of the ureter may be ruled out by catheterization The specimens collected will reveal similar findings since acute glomerulonephritis is a diffuse bilateral condition Phenol sulfonphthalein should be eliminated in relatively equal percen

tage from both sides. It is usual that the percentage elimination is lower than normal.

A diagnosis should not be difficult if the classical triad of symptoms of hematuria, edema, and hypertension is present. However, any one symptom may present a dominating feature, or the symptoms may be so mild as to be completely overlooked or even discounted. Mild forms of acute glomerulonephritis have been observed. In these mild forms a slight amount of albumin and a few red blood cells may be found in the urine. As time elapses, evidence of increasing intensity of the symptoms is demonstrated. Ultimately, a classical clinical picture of gross hematuria, hypertension, edema, and retention of nonprotein nitrogen is portrayed. Acute diffuse glomerulonephritis may begin with a fatal picture of uremia. Extensive albuminuria, gross hematuria, massive edema, hypertension, and a rapidly increasing nonprotein nitrogen retention in the blood may be observed. It is usual that the symptoms in the acute stage are less intense and may subside, recovery taking place.

Urinary findings: The volume of the urine is diminished in amount and is acid in reaction. The urine is of dark color and may present a smoky appearance. Albumin is present in excessive amounts. There may be sufficient blood to be observed macroscopically, or only microscopic amounts may be present. Microscopic examination of the urine sediment reveals hyalin or granular casts and great numbers of red and white blood cells.

Edema, when present, is more usually confined to the face, especially about the eyes. The edema may occasionally involve the hands, feet, and genitalia. It is considered as nephritic edema in contrast to the massive nephrotic edema of the second or subacute stage of the disease. The edema is due to increased capillary permeability. Recent investigative studies have shown that there is undoubtedly widespread vascular injury to the capillaries of the

subcutaneous tissue by the same process which affects the capillaries of the kidney in the acute stage Such vascular injury may permit an increased but generalized capillary permeability

Hypertension An elevation of blood pressure occurs in a high percentage of cases The elevation is moderate in degree Hypertension may suddenly occur and appear as the dominating symptom It is frequently associated with cerebral manifestations such as headache, convulsions and even coma These symptoms may be the result of a cerebral edema The mechanism of hypertension may be explained by the glomerular ischemia which accompanies the acute stage

Blood studies Chemical examination of the blood shows a moderate retention of nonprotein nitrogen An elevation of 35 to 50 mg per 100 cc of blood is usual The urea of the blood is also altered Normally, the urea volume is found to be about one half of the nonprotein nitrogen value In the acute stage of glomerulonephritis, it may be found to exceed 50 per cent of the nonprotein value Blood cholesterol may be moderately elevated during the acute stage

Renal function tests Urea clearance is the most helpful of all tests of renal function in the acute stage of glomerulonephritis Urea values of less than ten per cent of normal function may be seen Uremia is common when such a drop in the urea value occurs

Phenolsulfonphthalein excretion is not as sensitive a test as urea clearance and is therefore of less value When phenolsulfonphthalein is used the intravenous method of injection of the dye is to be preferred Peters and Van Slyke consider an excretion of 55 per cent or more in a two hour period as being within normal limits following the intravenous injection of the dye

Treatment For some inconceivable reason, the treatment of glomerulonephritis is considered to be the concern of the internist

rather than of the urologic surgeon. It frequently happens, too frequently in fact, that there simultaneously exists a "medical kidney" on one side and a "surgical kidney" on the other. The intelligent management of such a complicated condition is governed by the combined surgical and medical management before and after operative procedures are instituted. The urologic surgeon is frequently called upon by his medical confreres to perform surgical procedures in the presence of known renal disease, as well as to aid in differential diagnosis. It is believed that a thorough understanding of the management of the "medical side" of kidney disease is just as important to the urologic surgeon as the management of the "surgical side" of these organs in a diseased state.

There are several factors to be considered in the treatment of acute glomerulonephritis.

1. *Rest:* Rest in bed is imperative. Rest should be continued long after the acute symptoms of edema, hypertension, and hematuria have disappeared. The urine should show only the presence of a slight albuminuria or a few red cells before the patient is permitted out of bed. The most important factor of treatment is complete rest in bed, maintained regardless of age or of the sense of well-being portrayed by the patient.

2. *Diet:* The diet in the acute stage of glomerulonephritis is not a difficult one. The diet should be gradually increased as the general condition of the patient improves. Fruit juices in small amounts are permitted during the first few days following the acute onset of the disease. Cereals, junket, and custard may then be added to the diet. As improvement continues, the diet is increased by the addition of creamed vegetables, stewed fruit, eggs, and milk. Ultimately, a full diet, including meat, is permitted. Weiss considers the reduction of proteins necessary in only two phases of glomerulonephritis; at the beginning and at the end of the disease. . . . "At the beginning, only because one is dealing

with an acute infection in which heavy foods like meats would ordinarily be withheld; and at the end when renal failure occurs and the badly damaged kidney is having great difficulty in excreting, not only the end products of protein metabolism, but all other waste substances as well."

3. *Diuretics:* Diuretics are contraindicated. Stimulating diuretic action of drugs is harmful and should not be considered.

4. *Fluid intake:* Fluid intake should be limited in the acute stage of glomerulonephritis. At the onset, fluid intake should never exceed 1000 cc. in 24 hours An increase over that amount of fluid is determined by the ratio of the output of urine.

5. *Renal decapsulation:* Renal decapsulation is the stripping of the fibrous capsule from the kidney. Harrison, 1896, advocated the procedure. Decapsulation has been used extensively in the treatment of both acute and chronic nephritis with some degree of success in selected cases. It is very important, at the time of operation, to avoid leaving a remnant of the renal capsule attached to the kidney in such a fashion that it will later exert pressure upon, or strangulate the renal vessels. Such pressure or strangulation is capable of the production of what is now called a "Goldblatt kidney." Numerous hypotheses have been advanced as to the reasons for improvement following renal decapsulation. None of the theories has been too satisfactory in its explanation. De Takaats believes that improvement results from a partial sympathectomy with probable relief of vasospasm, rather than the release of the tension of congested, swollen kidney, compressed within a taut fibrous capsule.

Second Stage: Subacute Diffuse Glomerulonephritis (chronic parenchymatous nephritis)

This is the stage of degeneration and is characterized in its typical form by pronounced albuminuria and edema. The second

stage of glomerulonephritis progresses directly from the acute stage. In some instances, it is difficult to ascertain whether the condition present is a mild, acute attack or an active, subacute attack. It is possible that beginning as an acute attack of glomerulonephritis, the condition may pass through the second phase, to become chronic in a few months.

Clinically, the most predominate and characteristic feature of the subacute phase of glomerulonephritis is the presence of marked albuminuria and edema. The edema may be so extensive as to accumulate in the serous sacs, as well as the tissues. This nephrotic form of edema results from the depletion of blood protein caused by the continued loss of albumin in the urine. Another variety of the subacute form of glomerulonephritis may be observed. In this variety, hypertension is the most characteristic feature. In this variety, the hypertension is the most predominate symptom, while the edema is slight or entirely absent. Eventually, the subacute stage progresses to a chronic glomerulonephritis indistinguishable from nephrosclerosis.

Pathology: The gross appearance of the kidney in the subacute or second stage of glomerulonephritis is well described by the term, "large white kidney." The kidney so affected is enlarged and soft. The capsule strips easily and readily. The exposed surface is smooth and pale and has a slightly mottled appearance. The cut surface of the kidney shows marked swelling and pallor of the cortex. In contrast, the pyramids appear darker than normal. Yellow streaks or patches in the cortex may be observed. Microscopically, it may be observed that the glomeruli, tubules, and arteries, as well as the interstitial tissue, are involved (FIG. 320).

The glomeruli: Large numbers of glomeruli may show complete capillary occlusion and are filled by endothelial cells and proliferated hyalin material. Other glomeruli may show only partial occlusion. (The more extensive and widespread the occlu-

sion, the more quickly will the kidney pass into the atrophic or scarring stage) The glomerular capsules show definite changes There is an enlargement of the individual glomerulus The capsular space is filled in varying degrees by red blood cells desquamated epithelium albumin and fibrin

The tubules The degree and extent of tubular degeneration

Fig 370—Subacute diffuse glomerulonephritis Photomicrograph showing a later stage of crescent formation Note fibrosis of the tuft and hyalinization of the capsular proliferation.
(Courtesy of Dr Lawrence W Smith and Dr Edwin S Gault.)

correspond to the distribution and intensity of the lesions of the glomeruli If destruction of the glomerulus is complete, there will be a complete destruction of the tubules The epithelium of the tubules shows cloudy swelling, fatty degeneration, or necrosis As the tubular degeneration advances epithelial cells are cast off and the tubules become atrophic

Blood vessels The blood vessels during the subacute stage, begin to show the formation of hyalin material in the intima of

the veins and arterioles. The presence of hyalin material increases so that during the third or chronic stage it is apparent to a marked degree.

The interstitial tissue begins to show fibrosis with considerable fatty degeneration. It is too early in the progress of the disease to reveal the true characteristics found in the third stage.

Symptoms and Diagnosis: *Urine:* Albuminuria is more constant in the second stage of glomerulonephritis than in any other phase of the disease. The amount of albumin present may vary consid erably.

The quantity of urine voided is usually diminished. The specific gravity is usually increased. The specific gravity may vary, the level depending upon the amount of urine excreted and the degree of albumin present.

Hematuria: Hematuria is usually a constant finding as in the subacute stage of the disease. The amount of blood present may vary from microscopic quantities of red cells to an amount sufficient to be grossly observed.

Urine sediment: The centrifuged urine sediment reveals the presence of red blood cells, leukocytes, hyalin and granular casts. In addition, large "renal failure casts" may be found. These renal failure casts were originally described by Addis, who considered them of prognostic importance.

Edema: Edema is characteristic of the second phase of glomerulonephritis. It may be relatively slight in amount, affecting the defendent extremities of the body in varying degrees. It may be so massive as to fill the serous sacs of the body or even the tissues of the body. The second or subacute stage is the nephrotic or degenerative stage of glomerulonephritis. It is during this stage that albuminuria is the most marked. The albuminuria accounts for a great loss of plasma protein. The colloid osmotic pressure of the plasma is diminished, permitting the fluid to escape freely into the

tissues. Edema may be due to cardiac insufficiency as the result of an increasing hypertension and cardiac failure. The cardiac impairment interferes with the mechanism of the elimination of body fluids through the kidney. The kidney acts as a filter. Edema will result if permeability of the filter is disturbed or altered by any cause.

Blood changes. Anemia is a constant factor in the second or subacute stage of glomerulonephritis. The anemia is not as intense as is present in the third or chronic stage of the disease. The cause of the anemia is not known. It is possible that there is some interference in the formation of red cells or of hemoglobin. It is possible to evaluate the prognosis by noting the progress of this fall of the hemoglobin percentage.

There is an associated rise of the nonprotein nitrogen of the blood with renal insufficiency. Nonprotein nitrogen values of 100 mg. or more per 100 cc. of blood are frequently encountered. Blood urea nitrogen is moderate in the first stage, absent in the second stage, extreme in the third stage of glomerulonephritis.

Creatinine values often reach 10 to 20 mg. per 100 cc. In fatal cases, values as high as 28 mg. have been reported.

Plasma protein studies are important during the second or subacute stage It has been shown that the loss of serum albumin is largely responsible for the edema so evident in this phase of the disease. Edema is usually present when the total protein of the blood falls to 4.5 Gm. per cent. With this fall, there is reversal of the albumin globulin ratio Normally, this figure varies from 1.4 to 2.0, but in the presence of edema, this figure may fall as low as 0 6 Blood cholesterol is increased in nephritis with edema, and a definite rise is to be noted in the subacute stage of glomerulonephritis. It is a point of differentiation that while blood cholesterol values are high in nephritic edema, the values are normal in the edema of cardiac failure.

Hypertension: Moderate hypertension is to be noted in the acute stage, but usually assumes a normal range in the second or subacute stage. In the third stage, hypertension is one of the dominating factors of the clinical picture presented.

Uremia: Uremia is the usual fatal terminal phase of chronic glomerulonephritis. Mild manifestations may be observed in the subacute stage. Such symptoms as nausea, vomiting, muscular spasm, and stupor are occasionally seen.

Treatment: It is at this stage or phase of glomerulonephritis that edema is pronounced—so pronounced, in fact, that the clinical picture may closely simulate that of a true nephrosis.

As previously stated, the presence of edema is probably due to increased capillary permeability. A depletion of blood protein is occasioned by a continued loss of large amounts of albumin in the urine. This continued loss of protein must be overcome. The ingestion of protein foods must be increased. If the condition of the patient is such that a heavy diet is impossible, the intravenous administration of blood plasma or whole blood is indicated. The amount of protein administered must be greater than the continued loss, otherwise the patient is literally starved to death. The intravenous administration of whole blood or blood plasma is indicated in the presence of severe nausea and vomiting, extreme abdominal distention, lowered urinary output with an increasing retention of blood urea nitrogen. A transfusion of 500 cc. of whole blood may be given every second day. Cardiac impairment or other complications are treated if and when they occur. As the severity of the symptoms subsides, the maintenance of a diet becomes the paramount question of management. As in any other acute condition, fruit juices are permitted for the first few days, then cereals, junket, and custards are added. Creamed vegetables and stewed fruits, in addition to milk and eggs, are added as the

natural demand for food increases Finally a full dietary regime, including meat, is established

O'Hare and Vichers use the following simple, yet complete and efficient outline in the determination of the quantity of protein in the diet It is given verbatim

Any combination of the foods listed below may be selected

Foods not listed below must not be taken

In Groups I and II there is a restriction in the total amount

The foods in these groups must be served in full or half portions

A full portion in Group I counts 1

A full portion in Group II counts 2

In Group III the quantity of each is not restricted, although you are urged to use discretion

Points on recipes to count as indicated

Your total score for the day should be

Your total amount of fluid should be pints Do not add salt or spices to the food after it has been cooked

GROUP I

(Each full portion counts 1)

	Full Portion	*Vegetables, etc*	*Full Portion*
Bread (white)	1 av slice	Baked beans	1 tablespoon
Bread (graham)	1 av slice	Lima beans	1½ tablespoons
Uneeda biscuit	5 crackers	Potato creamed	1 tablespoon
Shredded wheat	1 biscuit	Potato mashed	1½ tablespoons
Graham crackers	5 crackers	Potato baked	1½ medium
Cereals etc		Potato boiled	1½ medium
		Canned corn	2½ tablespoons
Oatmeal	2 tablespoons	Green peas	2 tablespoons
Boiled rice	3 tablespoons	Beets	5 tablespoons
Cornmeal mush	4 tablespoons	Spinach	4 tablespoons
Cream of wheat	6 tablespoons	Bananas	2 large
Farina	6 tablespoons	Cream heavy	⅔ cup
Macaroni	1½ tablespoons		

GROUP II

(Each full portion counts 2)

	Full Portion
Milk	1 glass
Egg	1 egg
Eggs (scrambled)	1½ tablespoons
Flour, sifted	⅔ cup

Meats

Lamb chop, broiled	⅔ chop
Lamb roast	3" x 2½" x ¼"
Beef, roast	3" x 2" x ¼"
Beef steak, broiled	2" x 1" x 1"
Chicken, roast	3" x 3" x ⅛"

Fish	*Full Portion*
Cod, boiled	1" x 1" x 1½"
Haddock, boiled	1" x 1" x 1½"
Halibut, boiled	1" x 1" x 1½"
Mackerel, boiled	1" x 1" x 1½"
Salmon, boiled	1" x 1" x 1½"
Smelt	½" x 1" x 3"
Oysters	7 oysters
Crabmeat, canned	2 tablespoons
Salmon, canned	1½ tablespoons
Shrimp, canned	6 small

GROUP III

(No restriction)

Vegetables	*Fruit*	*Miscellaneous*
Asparagus	Apple	Sugar
Cabbage	Apricot	Maple sugar
Carrots	Blueberries	Syrup
Cauliflower	Cherries	Honey
Celery	Cranberries	Candy
Cucumbers	Grapefruit	4 dates a day
Lettuce	Grapes	3 Sunshine arrowroot cookies a day
Mushrooms	Muskmelon	Cornstarch
String beans	Lemons	Arrowroot
Tomato (fresh)	Oranges	Tapioca
Tomato (cooked)	Peaches	Post Toasties
Onions	Pears	Butter
Squash	Pineapple	Olive Oil
Turnips	Plums	
	Prunes	
	Raspberries	
	Strawberries	
	Watermelon	

"On this sheet are most of the ordinary foodstuffs used in any home. These foods are divided into three groups according to the amount of protein in them. In Group III, there is so little protein that one can ordinarily ignore it. In Group I, each full portion (indicated to the right of each foodstuff) contains approximately 4 grams of protein. In Group II, each full portion contains approximately 8 grams of protein. To make it still simpler, we avoid mentioning grams and instruct the patient that each full portion in Group I counts one point. In Group II, it counts two points. The number of points for the day is inserted in the blank space left for that purpose at the top of the sheet. A low protein diet would be represented by seven points (28 grams). A very generous protein diet—for a nephritic patient—would be equivalent to fifteen points (60 grams). Another blank line for the prescription of the amount of fluid is placed immediately under the prescription of protein. The physician may use his own discretion about allowing tea or coffee or substitutes for these. The patients are not allowed to add salt to the food after it comes to the table. The average patient under this regime does not get more than 4 or 5 grams of salt a day. If he is edematous, we can still further decrease the salt by ordering fresh butter, salt-poor bread, vegetables, meats, etc., boiled free from salt and, if necessary, the use of distilled water whenever water is used in the diet. The former method reduces the salt intake to approximately 2.0 grams a day, and the latter to perhaps as low as 0 5 grams a day."

There are several other factors that should be observed and regulated during the maintained dietary regime.

A careful check of the fluid intake and output is maintained throughout the entire time that the clinical picture is being unfolded Repeated observation of the specific gravity of the voided urine should be made. A reading of the specific gravity level made at the time of each voiding will afford a true indicator of the con-

centrating ability of the kidneys. It is usual that the specific gravity of the urine during the subacute stage of glomerulonephritis will be maintained from 1.002 to 1.010, the urine output ranging in the neighborhood of 3000 cc. (100 ounces) during a 24-hour period. Elimination of waste products is thus maintained by a urine of low specific gravity, but great in volume. This is in contrast to the normal elimination of urine of a higher specific gravity but less in amount. As elimination of waste products is maintained and continued, a decrease in the retention of the non-protein nitrogen of the blood will be noted. The rapidity of this decline depends upon the extent to which the kidneys are able to rehabilitate themselves to normal excretory action.

Diuretics: Diuretics may be continuously employed in the edema of glomerulonephritis. In fact, the closer the clinical picture simulates a true nephrosis, the safer the administration of diuretics. Strict attention should be paid to the possibility of reactions to such drugs. Should such reactions occur, immediate discontinuance of the drugs is indicated.

Third, or Chronic, Stage of Glomerulonephritis

The chronic stage of glomerulonephritis is the stage of scarring. Such scarring results from the kidney having been subjected to the ravages of inflammation through the acute and subacute stages of the disease. Frequently, the first or acute stage may have been so mild as to escape detection and to pass ultimately into the second or subacute stage. There is no evidence that would point to the fact that the third stage of glomerulonephritis begins of itself and is not the aftermath of an acute stage. Once established, chronic glomerulonephritis is certain to end in renal insufficiency and uremia. The renal insufficiency so apparent in this stage is the result of a gradual transformation of the kidney parenchyma into scar tissue.

Pathology The gross appearance of the kidney depends on the severity of the scarring process The kidney is small and shriveled The external surface is rough and finely granular The renal capsule strips with difficulty and is usually so adherent that portions of the cortex tear away with it The cut surface is extremely irregular and reveals an atrophy of the cortex The medium sized

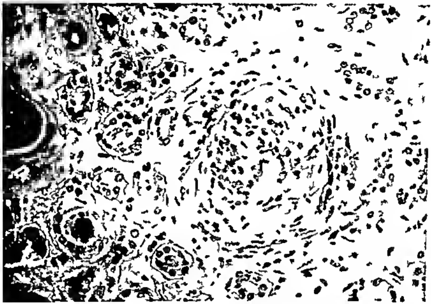


Fig 321—Chronic glomerulonephritis Highpower photomicrograph showing almost complete replacement of glomerulous by fibrotic tissue The capsular space is likewise almost completely obliterated.
(Courtesy of Dr Lawrence W Smith and Dr Edwin S Gault)

vessels are thickened and sclerotic in appearance Microscopic examination reveals an almost complete loss of renal structure (Fig 321) The tubules are largely replaced by fibrous tissue and the glomeruli show marked hyalinization

Symptoms The disease may be far advanced before it is observed in some patients even though renal efficiency is so impaired that uremia is imminent The symptoms are usually

insidiously progressive. Individually, the usual symptoms may be considered as:

Hypertension: Hypertension is a dominating feature of the third or chronic stage of glomerulonephritis. The blood pressure usually remains below 200 mm. The hypertension is apparently a compensatory mechanism by which the glomerular pressure is raised. This increased glomerular pressure causes a certain degree of function of those glomeruli that are not completely fibrosed Without this compensatory filtration, uremia would become apparent more quickly in the majority of instances.

Urinary changes: There is marked impairment of renal efficiency as revealed by the functional tests. The amount of urine excreted is increased, but the specific gravity is lowered and at a fixed level. There is a failure to concentrate above 1.010. Fishberg states that . . . "impairment of renal function involves all of the excretory functions of the kidney. No matter what the anatomic substrations—arteriosclerotic or inflammatory changes, polycystic transformation or prostatic obstructions—impairment of renal function is always manifested in the same way; namely, by a lowering of the maximum concentration in which each of the individual urinary constituents can be excreted. As the impairment of renal function progresses, the maximum specific gravity attainable falls correspondingly."

The individual survives as the result of quantity elimination rather than quality elimination of urine. Polyuria may be the only symptom present. This symptom is attributed by the patient to the fact that there is present an unquenchable thirst. The polyuria may be manifested only by an extreme nocturia. The amount of urine voided at night may approach or even exceed the amount of urine voided during the day. This nocturnal voiding may be caused by many factors other than kidney disease. Nocturia

should be disregarded as a nephritic symptom if the urine is clear and renal function unimpaired.

Albuminuria: Albuminuria is present in the third or chronic stage. Its presence results from interference with the renal filter, particularly the glomeruli. The urinary sediment contains hyaline and granular casts, leukocytes, and a limited number of red blood cells.

Blood changes: Anemia is a constant symptom at this stage. The cause is not known. Peters and Van Slyke have shown that the degree of anemia present is proportionate to the degree of renal insufficiency.

Nonprotein nitrogen retention in the blood may be extreme. Such retention may be 100 mg. or more per 100 cc. of blood. Creatinine retention is also evident and may rise to 20 mg. or higher per 100 cc. of blood. There is a diminution of serum protein and a reversal of the albumin globulin ratio.

Renal function tests: The phenolsulfonphthalein test reveals a marked diminution in excretory ability. Occasionally, a reading as low as ten per cent for a two hour total may be recorded. Urea clearance is much below normal. The Mosenthal concentration test reveals a specific gravity fixed below 1.015.

Edema may be evident, but is never so extreme as is seen in the second stage. An associated cardiac edema may exist which is evidenced by decompensating cardiac impairment.

Treatment: There is no specific treatment because the chronic stage of glomerulonephritis is progressively fatal. All efforts should be directed to the maintenance of nutrition and to provide sufficient fluids to accommodate the compensatory polyuria.

UREMIA

Uremia is an intoxication induced by renal failure and characterized by marked nitrogen retention in the blood. Uremia is

usually a complication of nephritis. It may, however, result from the suppression of urine of obstructive uropathy, such as bilateral impacted renal or ureteral calculi, or prostatic hypertrophy (FIG. 322). Nitrogen retention is invariably present, but it is not the

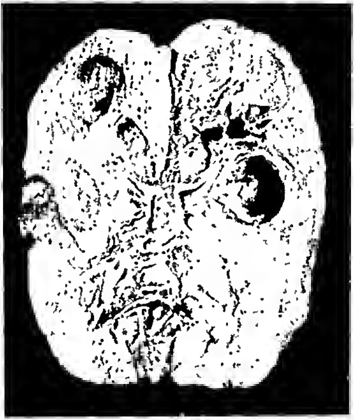


Fig. 322—Leukemic infiltration of kidney. An unusual cause of uremia. Note uniform appearance of parenchyma and lack of normal renal characteristics.
(*Temple University Hospital, Acc. No. 3433*)

cause of uremia, yet it serves as a valuable and accurate index of its presence. Uremia may be acute or chronic. There are several classifications of the acute type according to different manifestations:

1. *Cerebral,* with excitement, apathy, forgetfulness, muscular

twitchings, delirium, convulsions and coma The mechanism producing convulsions is not known They cannot be explained wholly on the basis of hypertension Cantaron has shown that when muscular twitching is evident in the presence of chronic nephritis, there is a diminution of the diffusible calcium of spinal fluid It may be that the convulsions result in simultaneous existence of diminished calcium and the existing hypertension The cerebral type of uremia is the one most frequently seen in acute nephritis

2 *Gastrointestinal,* with urinous odor to the breath, anorexia, nausea vomiting, abdominal distention, and diarrhea This symptom complex is usually seen in chronic nephritis

3 *Pulmonary,* with paroxysmal dyspnea

Regardless of the type portrayed, uremia is evidently a toxemia produced by a toxin or toxins, the nature of which is not known

Treatment Uremia, in its many different manifestations, may respond quickly to treatment Intravenous injections of five to ten per cent glucose in normal saline will aid materially, particularly where dehydration has been induced by vomiting Administration of blood plasma or transfusions of whole blood may be given to maintain a more normal blood protein content The loss of serum protein is constant in albuminuria

Chloral hydrate or an opium derivative may be administered to control the acute, convulsive stage of uremia Lumbar puncture will frequently stop convulsive seizures, affording temporary relief of the symptoms In the acute convulsions of uremia the use of intravenous fluids should be carefully watched It has been proven that during such a time, there is edema of the brain Any addition of fluid will increase the cerebral edema and the convulsions will be intensified The desired effect of increased kidney elimination will not occur

Chronic Uremia

It is this form of uremia that is of considerable concern to urologists. It is frequently observed in prostatic obstruction of long standing. This type of uremia seldom develops except in instances of chronic yet extensive impairment of kidney function. The onset is insidious and may be continued for months without apparent effect.

The symptoms portrayed are less intense in the early stage of chronic uremia than those observed in the more acute types. The tongue becomes dry and apparent furrows develop on a thick, brownish-white coating. The edges of the tongue appear red in contrast to the thick, whitish coating. The skin becomes dry and scales readily. In instances of extreme loss of weight, the skin appears tautly stretched over the bones and joints. There is a lack of desire for food; even the most bland foods may be regurgitated. The desire for fluids is lost. The breath becomes heavy and foul, presenting the characteristic urinous odor. As the uremia progresses and becomes more profound, the digestive disturbances and bowel irregularities are intensified. Hiccough is frequently constant, resisting all measures to counteract it. Finally, stupor develops and coma slowly supervenes, with typical Cheyne-Stokes type of breathing. As the stupor develops, marked irritability and periods of excitement may be observed. Vesical disturbances are nearly always present. Albuminuria, retinitis, and vascular changes in the retina are frequently seen.

The blood shows marked retention of urea nitrogen, from 75 to 150 mg. per 100 cc. of blood is not uncommon. Creatinine shows a marked increase; 5 to 30 mg. per 100 cc. are not infrequent.

The picture portrayed is a slowly progressive, downward decline and a steadily increasing retention of waste products as the renal insufficiency continues.

Treatment Treatment of uremia should be (1) The promotion of an abundant elimination of urine, (2) the maintenance of micturition, (3) general care Relief of the obstructive uropathy, if possible, should be undertaken In the event of prostatic enlargement, catheter drainage or suprapubic drainage may be maintained over a period of months if the patient's physical condition prevents major surgical procedure Frequently, it is possible to relieve the advancing uremia and promote a physical state permitting the surgical removal of the obstructing prostate or other obstructive uropathy

Diet The proteins may be restricted but not prohibited Restriction of proteins to a daily consumption of 30 to 40 Gm should be made Carbohydrates in adequate amounts, to balance the proteins, should be permitted Vomiting may be present to such a degree that the suggestion of food or fluid may cause continuous regurgitation The vomiting leads to dehydration and starvation by the continuous loss of fluid and food and causes the nonprotein nitrogen of the blood to mount rapidly Because of the vomiting it may be necessary to stop all nourishment by mouth and to give fluids by other channels

Intravenous administration of five per cent glucose in saline, blood plasma, or whole blood transfusions may temporarily check the downward course The intravenous administration of fluids should be given very slowly, never faster than 200 cc per hour

Occasionally, the progressive decline may be checked Usually, however, once firmly established, chronic uremia continues its downward course in spite of all measures to forestall it

NEPHROSIS

Nephrosis is a degenerative lesion affecting principally the renal tubules The clinical picture presented is one of massive edema

and albuminuria, associated with alteration of the blood chemistry. A very similar picture is presented in the second stage of glomerulonephritis. Boyd, in discussing the subject states . . "that chronic nephrosis is merely the second stage of glomerulonephritis, in which the glomerular lesions may be minimal, but sufficient to damage the renal filter and allow continued escape of a large quantity of protein. The other features of the complex—edema, low plasma protein, reversal of the albumin-globulin ratio, hypercholesterolemia, low basal metabolic rate—may all be secondary to this loss."

Fishberg believes that lipoid nephrosis exists as a clinical entity. He considers that one of the most important differential diagnostic points in glomerulonephritis is the complete absence, or the presence of a minimal number of red cells found in nephrosis, as contrasted to varying degrees of hematuria observed in glomerulonephritis. Lipoid nephrosis (Epstein), genuine nephrosis (Volhard & Fahr), or chronic nephrosis, if it actually exists, must be differentiated from glomerulonephritis. It is not to be confused with amyloid disease of the kidney, the chemical nephrosis of bichloride of mercury poisoning, or the toxic nephrosis of pregnancy. These three latter conditions exist, but do not present a confusing or questionable overlapping of the clinical picture or of the pathological findings.

Pathology: The appearance of the kidney is grossly one of a "large, white kidney." The kidney is somewhat enlarged, the capsule stripping readily, exposing a smooth, pale surface. The cut surface of the cortex is pale in comparison to the dark pyramids. In the cortex are bright yellow streaks or patches due to a deposition of lipoid material. Microscopically, the cells of the convoluted tubules are swollen and the cell body may be filled with lipoid material. In staining, a fat solvent is used, the cells pre-

senting a vacuolated appearance The glomerular capillaries show a proliferation of the endothelial lining and their basement membrane shows thickening Identical pathological findings are observed in the second stage of glomerulonephritis

Symptoms and Diagnosis: The onset of lipoid nephrosis is insidious It is usually rather far advanced when first observed The most characteristic symptoms are those of marked edema and massive albuminuria The urine is of high specific gravity, showing many casts but no red cells There is a sharp increase of blood cholesterol, which may rise to 700 to 800 mg per 100 cc There is no evidence of retention of blood urea nitrogen, the blood pressure is normal but there is a moderate but progressive anemia The anemia probably results from under nutrition rather than indicating definite thyroid disease The basal metabolic rate is low General symptoms such as headache, malaise and anorexia may accompany the onset of the edema Loss of weight is common but is often masked by the massive edema When the edema is controlled, the loss of weight will become evident Renal insufficiency and uremia do not occur The condition runs a chronic course with remissions The patient is predisposed to infections, notably pneumococci peritonitis, which may be fatal In children the condition is frequently found associated with sinusitis

Cystoscopy There are no pathognomonic cystoscopic characteristics of lipoid nephrosis There is usually no impairment of renal function as revealed by dye elimination tests A relative urinary output is maintained

AMYLOID NEPHROSIS

Amyloid nephrosis of the kidney results from advanced tuberculosis, chronic suppuration, or syphilitic lesions Some investi

gators believe that amyloid disease should always be considered in chronic pulmonary tuberculosis with suppurative complications in which albuminuria, casts, and edema occur. The amyloid material is deposited in the glomeruli of the kidney. It also involves the walls of the arterioles with stoppage of the circulation through the glomeruli. In this way, there is an interference of renal function which ultimately may account for renal insufficiency. As a result of obstruction to the glomerular circulation, the convoluted tubules may gradually atrophy. The tubules are replaced by fibrous tissue. The shrinking of the fibrous tissue produces a contracted kidney similar to that of chronic nephrosclerosis.

NEPHROSCLEROSIS

Essential hypertension is accompanied by degenerative, fibrotic, and sclerotic changes in the kidneys known as nephrosclerosis (FIG. 323) Nephrosclerosis may be divided into two groups: (1) Benign nephrosclerosis is a renal sclerosis with benign hypertension and little or no renal insufficiency; (2) malignant nephrosclerosis is a renal sclerosis with malignant hypertension and marked renal insufficiency. Both conditions show hypertension but the renal changes vary.

BENIGN NEPHROSCLEROSIS

In the benign form, the patient may live for many years without signs or symptoms of renal insufficiency. Sooner or later the renal impairment will be revealed by a lowering of the specific gravity of the urine, the presence of a slight amount of albumin and the occasional presence of granular or hyalin casts. Glomerular sclerosis may continue for years. A cerebral vascular accident or cardiac impairment resulting from the hypertension usually

intervenes before complete renal insufficiency and uremia develop

MALIGNANT NEPHROSCLEROSIS

Relatively ten per cent of all cases of hypertension develop serious impairment of renal function Development of renal failure may occur after short or long periods of benign hypertension The power of urine concentration by the kidney is rapidly lost The specific gravity of the urine becomes stationary at a relatively low figure of 1 010 or below Albuminuria is present with the associated presence of casts and red blood cells A rapidly progressive retention of nonprotein nitrogen of the blood is present Uremia ensues with the rapidly advancing renal insufficiency

THE SENILE ARTERIOSCLEROTIC KIDNEY

This form of nephrosclerosis is only a part of a generalized arteriosclerosis There is neither hypertension nor renal insufficiency present The kidney is contracted and scarred, which is the result of infarcts induced by marked atheromatous changes in the aorta and renal artery The renal artery may be so markedly atheromatous that the lumen may be distinctly narrowed Interspaced between these areas of fibrosis and scarring, normal secreting parenchyma exist Albutt describes it as a "starved but not a corrupt kidney, sufficient for the smaller life of an elderly man The condition does not result from essential hypertension, because what remains of the parenchyma excretes normally so that there is little or no danger of renal failure

MERCURY NEPHROSIS

Mercuric bichloride poisoning produces a very destructive variety of nephrosis Besides the renal lesions, gastrointestinal

ulcerations are common. Regardless of the mode of entry into the body by the mercury salt (skin, vaginal mucous membrane, or mouth), it is passed into the large bowel, producing an intense hemorrhagic colitis. It is also excreted by the kidney with the pro-

Fig. 323—Kidney—Nephrosclerosis, arteriolar type (essential benign hypertension). Lowpower photomicrograph showing a small renal arteriole, the site of concentric hyperplastic sclerosis. Note similar changes with obliteration of arterioles, interstitial fibrosis and glomerular scarring.

(Courtesy of Dr. Lawrence W. Smith and Dr Edwin S Gault)

duction of characteristic phenomena, anuria and marked nonprotein nitrogen retention. Although death may occur from renal failure (uremia), in many cases it is due to the extraurinary lesions produced by the poisonous agent. Edema is conspicuous by its absence.

Pathology: The renal lesion is confined to the tubular epithelium where necrosis with calcification takes place. The extent of the renal lesion is in direct ratio to the amount and concentration

of the mercury delivered to the kidney through the blood stream (FIG 324) For this reason the condition is bilateral both kidneys being uniformly affected The renal lesions vary from mild congestion to extensive necrosis with calcification and coincident urinary suppression and anuria The acute calcification occurs

Fig 324—Kidney—Acute toxic nephrosis (bichloride poisoning) Photomicrograph in which advanced tubular degeneration and necrosis are seen Note distribution especially to convoluted tubules.
(Courtesy of Dr Lawrence W Smith and Dr Edwin S Gault)

within a period of a few hours the calcium salts are deposited in the masses of necrotic cells within the tubules As these pathological processes continue anuria becomes complete There is also a continuously advancing destruction of body protein There is a loss of sodium chloride caused by vomiting Dehydration is induced by vomiting and by diarrhea These factors contribute to the production of extreme nonprotein nitrogen retention in the blood

Symptoms and Diagnosis: The ingestion of bichloride of mercury is usually immediately followed by vomiting and diarrhea. The patient may succumb to a condition resembling surgical shock before any renal damage is demonstrable. The majority of patients recover without evidence of renal damage, due to the fact that the vomiting is so immediate that absorption of the poison never occurs. In those instances where absorption has occurred, the symptomatology is characteristic: Anuria, complete and remaining so, regardless of all efforts to restore urinary secretion, vomiting and diarrhea; and a continuously advancing retention of blood nonprotein nitrogen are evident. Uremia and ultimately death ensue.

Cystoscopy: There are no pathognomonic features. Anuria being complete, there is no evidence of kidney function as revealed by dye function tests. Ureteral catheterization reveals a normal patulousness of the ureters. Pyelography is definitely contraindicated but, if done, it would reveal a normal kidney pelvis and calices. Intravenous urography is useless due to complete lack of urinary excretion.

Treatment: Rosenthal, 1934, advocated the use of sodium formaldehyde sulfoxylate as the specific antidote for acute mercury poisoning. The procedure of administration, as originally suggested, is: Immediate gastric lavage by stomach tube using a five per cent solution, permitting 200 cc. to remain in the stomach after completion of the lavage. An intravenous injection of ten Gm. of the salt dissolved in 100 to 200 cc. of distilled water should be administered slowly, taking 20 to 30 minutes. After six hours, intravenous injection may be repeated, using 0.1 Gm. of sulfoxylate per kilogram or a total of five Gm. for an adult of average weight. High colonic irrigations with a 1:1000 solution of sulfoxylate may be used once or twice a day.

The important factors influencing the outcome of such a regime

are The amount of the poison ingested, the amount vomited the amount absorbed and the promptness with which the antidote is administered It is believed that the damage to the renal tissues is complete within a few hours after ingestion of mercuric bichloride, that the administration of sulfoxylate solution is useless after two or more hours

Bilateral decapsulation has been suggested, but as yet no statistical data are available as to the efficiency of such a procedure

RENAL RICKETS

Renal rickets is a form of dwarfism or stunted body development in association with and probably resulting from renal insufficiency

In 1883, Lucas attracted considerable attention to the subject He found that the presence of albuminuria occurred in certain types of rickets and called the disease rickets of adolescence Fletcher and also Parsons (1911) were able to demonstrate the relationship between renal insufficiency in childhood and skeletal bone changes Barber, 1911, who has written extensively on the subject and to whom credit is given for the establishment of the condition as a clinical entity, defined it as a condition of stunted development, associated bone deformities of the late rickets type due to an insidious chronic interstitial nephritis of obscure etiology

The relationship between the rachitic bone changes and renal insufficiency is obscure Renal rickets is usually observed in late childhood but beyond question, the renal insufficiency dates from birth or from early infancy There are no specific forms of renal dysfunction in which the condition is allied but it is observed in the presence of chronic nephritis or bilateral obstructive uropathy where there is a gradual developing and advancing renal

insufficiency. As Greene has pointed out, it is not the pathological changes in the kidney, but rather the functional renal insufficiency, regardless of its cause, that determines the clinical picture. However, Price and Davie believe that renal rickets is a syndrome common to the two separate and distinct diseases, one the late sequelae of hyperparathyroidism; and the other, renal insufficiency.

It may thus be seen that the cause of renal rickets is obscure and undetermined. There are several theories, but none can fully explain the clinical picture presented.

1. Renal theory, in which there is a clinical deficiency resulting from renal insufficiency regardless of its cause.

2. Hyperparathyroid theory, in which there is parathyroid hypertrophy secondary to renal insufficiency.

3. Pituitary diencephalon theory, in which both the pituitary and the diencephalon are diseased and the syndrome presented results from a combination of the features of both diseased structures.

Out of all this haze of uncertainty stands a stunted, dwarfed child with definite progressive renal insufficiency, whose life span is short and whose chance of cure is remote.

Symptoms and Diagnosis: The condition begins undoubtedly at birth. It is seldom recognized until after the age of two years, when retarded body growth may be noted. It is not until the seventh or eighth year that the development of bone deformities may be noted. *Either sex may be affected. The skin is dry and is occasionally pigmented.* The child is short in stature and walks with a waddling gait, which varies in extent with the degree of bone deformity, of which knock knee (*genu valgum*), is the most common. The urine is of low fixed specific gravity and is increased in amount. Polydipsia, polyuria, and nocturia are common. As the disease progresses, anorexia, headache, nausea, and vomiting are

usually manifest. An estimation of blood calcium and blood phosphorus is important. Normally, in children, the blood phosphorus is 3.5 to 5 mg. and blood calcium is 9 to 11 mg. per 100 cc. In the usual type of rickets, the blood calcium increases while blood phosphorus decreases. In renal rickets, the opposite is true; the blood calcium decreases while the blood phosphorus increases. This difference of ratio is a highly significant finding in differential diagnosis. As the renal insufficiency increases, a gradual rise of nitrogenous substances in the blood and a gradual fall in phenolsulfonphthalein output will be noted. Hypertension is uncommon until the terminal stage, which is usually uremia.

Cystoscopy: Cystoscopy, if done early, may contribute a great deal in ascertaining the presence of obstructive uropathy. As has been pointed out, any obstructive uropathy may be the fundamental cause of early renal insufficiency. If, by cystoscopy, an obstructive lesion such as vesical neck obstruction or valves of the posterior urethra, may be found, much may be done to improve the renal insufficiency. Likewise, it is also possible, by cystoscopy, to ascertain accurately the degree of renal insufficiency of both kidneys.

Treatment: Little can be offered in the treatment of renal rickets. The relief of urinary obstruction, combating the renal insufficiency, and the associated acidosis, are the salient factors of treatment.

THROMBOSIS OF THE RENAL ARTERY

(atherothrombosis of the renal artery)

The analogous similarity between thrombosis of the renal artery and coronary thrombosis is emphasized by Wolffe. Thrombosis of the renal artery is not to be confused with embolism, although the clinical manifestations may be quite similar. Embolism of the renal artery is an accident resulting from a disease process else-

where in the body. The origin of such an embolus is generally the heart, as the result of a vegetative endocarditis. Thrombosis of the renal artery may be considered as a local manifestation of a generalized atheromatosis.

Symptoms and Diagnosis: *Pain:* Pain in the renal area is a most constant symptom. The pain is localized in the renal area and may simulate renal colic in its acuteness and intensity. The pain may be dull but continuous.

Tenderness over the affected kidney is invariably present.

Rigidity of the lumbar muscles is usually present.

Urinary findings: Immediately following the onset, frequency of urination may be experienced by the patient. There is usually an increased amount of urine excreted which accounts for the frequency of urination. Following the temporary polyuria there is a diminution in the amount of voided urine. Hematuria, in varying degrees, is a constant finding. The amount of blood varies from a few red cells observed microscopically, to gross hematuria.

Systemic symptoms are common. Vomiting, headache, abdominal distention, and at times, shock may be observed. Abdominal distention may present unusual and alarming proportions. Distention may be so intense as to simulate paralytic ileus. General physical findings point to the presence of a preëxistent generalized, atheromatous, cardiovascular disease.

Cystoscopy: There is nothing pathognomonic in the cystoscopic picture presented. Individual function tests may show a diminution of percentage elimination of the affected side. If total occlusion of the renal artery has occurred, the affected side will be functionless.

Roentgenological findings are suggestive rather than absolute. Retrograde pyelography does not reveal any abnormalities. The pyelogram is normal unless there is some associated urological

lesion. Intravenous urography may reveal a shaggy, marginated defect within the general outline of the kidney.

Treatment: Absolute bed rest is essential. Immediately following the onset, papaverine hydrochloride may be given intravenously or orally in 0.03 to 0.06 Gm. (one-half to one grain) doses every three hours. As the acuteness of the condition subsides, the drug may be continued, orally administered, for several weeks.

ANEURYSM

True aneurysm of the renal artery is rare. The condition was first described in 1791. Since that time, less than 80 cases have been reported, many of which have not been proven conclusively.

The cause of spontaneous aneurysm is not known although Mathé considers trauma as the most common etiological factor. Many of the cases reported did not present a history of injury but arose because of some primary disease of the blood vessels. Arteriosclerosis, inflammatory changes of the arterial wall, periarteritis nodosa, and syphilis are frequently considered as causative factors (FIG. 325).

Symptoms: The symptoms of aneurysm are pain and hematuria. The pain may be intermittent with prolonged free intervals. Hematuria is usually recurrent and may be initiated with the slightest exertion. The aneurysm may rupture into the renal pelvis or about the kidney producing acute symptoms of pain or hematuria. The hemorrhage following rupture may be so extensive as to produce a rapidly enlarging tumor of the loin, sudden weakness, and the anemia of hemorrhage. The classical signs of aneurysm such as pulsation, bruits, and murmurs are seldom present.

Diagnosis: The diagnosis of true aneurysm, if uncalcified, is im-

possible before operation or until it ruptures. If calcified, the "wreath" of a rounded or fusiform shadow with light center and dense periphery is quite characteristic but must be differentiated from calcified lymphatic glands, renal calculi or gall stones.

Fig. 325—Aneurysm of renal artery.
(Temple University Hospital, Acc. No. 1634.)

Cystoscopy: There is no pathognomonic cystoscopic finding. If hematuria is evident, the appearance of blood from the ureteral orifice is not characteristic.

Treatment: Nephrectomy is the only treatment.

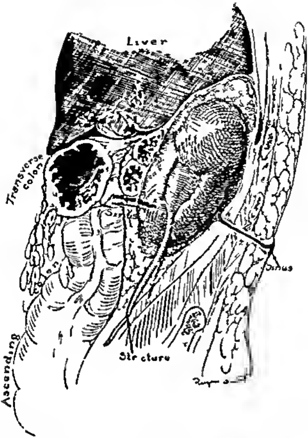


Fig 326—Perirenal abscess with resultant renocolic fistula external sinus and ureteral stricture A loop of duodenum lies between the colon and the kidney directly above the sinus.

(Courtesy of Dr Miley B Wesson and *Journal of Urology* 1938 39 589)

RENOINTESTINAL FISTULÆ

A fistulous tract existing between a kidney and the bowel is not uncommon Renointestinal fistulae are secondary to a chronic inflammatory lesion of the kidney The inflammation is associated with a perinephritis or perinephric abscess The kidney may rupture directly into the intestine or indirectly through an abscess The ultimate rupture of the kidney or perinephric abscess may be into the colon or the duodenum (Fig 326) The in

flamed kidney or walled-off abscess becomes attached to the colon or duodenum by fibrous adhesions. When perforation takes place, the fever subsides and the perinephric tumor disappears as the pus is evacuated through the newly created fistulous tract (FIG. 327).

Symptoms: The symptoms of renointestinal fistulae vary according to the position of the perforation. Gastrointestinal symptoms are evident in those instances in which the perforation exists with the duodenum. Weight loss and anemia are common. The preceding perinephritis or perinephric abscess presents the classical symptoms of chills, fever, and occasionally a palpable tumor in

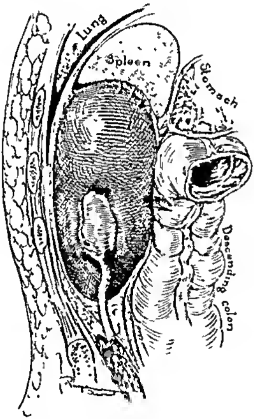


Fig. 327—Left renocolic fistula, large walled-off abscess in upper pole of kidney.
(Courtesy of Dr. Miley B. Wesson and *Journal of Urology*, 1938, 39,559.)

the loin may be elicited. When a fistulous tract exists directly between the kidney and the colon, continuous dull pain may be experienced. In the presence of any fistulous tract communicating with the kidney or renal pelvis, pyuria is a constant finding Hematuria, in microscopic or in gross amounts, may be present.

Cystoscopy· There is nothing pathognomonic of fistulous tract formation referable to the bladder. It is usual that an inflammation of varying intensity is present The ureteral orifice of the affected side may be markedly inflamed and edematous, as would be observed in acute infection of the upper urinary tract. The

Fig 328—Reno-gallbladder fistula Note the wide distribution of the radiopaque medium

(Courtesy of Dr G P Gambalvo Temple University Hospital, Acc No 25465)

ureteral urinė may show varying numbers of pus cells. None of the findings is characteristic.

The greatest diagnostic finding is to be obtained by retrograde pyelography. The sinus and the organs to which the sinus extends may be outlined by the opaque medium (FIG. 328).

Treatment: Nephrectomy is indicated, with dissection of the fistulous tract. Closure of the distal end of the sinus is imperative.

XXIV

SPECIFIC INFECTIONS OF THE KIDNEY

URINARY TUBERCULOSIS

Renal tuberculosis is one of the most interesting and important diseases affecting the genitourinary system. Renal tuberculosis is usually associated with or followed by ureteral and vesical involvement. The disease is considered in its relationship to all three organs (PLATE LXX).

The variations, changes, and ramifications of genitourinary tuberculosis are such that the progress of the disease is unpredictable. Being so unpredictable, vital interest is maintained clinically, cystoscopically, operatively, and pathologically. Because of all the varied changes portrayed by this condition, the cystoscopist is called upon to exert his greatest skill in manipulation and interpretative ability of the lesions present. Cystoscopy and cystoscopic manipulation may be difficult in advanced chronic renal tuberculosis with secondary contraction and irritability of the bladder. Skill and gentleness of technic are basic keynotes to be carefully observed in spite of anesthesia or analgesia. Trauma, added to the already present infection, may be the causative factor in making life, which is already uncomfortable, one of abject misery for the patient.

Renal tuberculosis is a secondary tuberculous lesion. Of all the tuberculous lesions of the urogenital system, renal tuberculosis

Examination

Tumor

Usually no evidence of *enlarged kidney. Tenderness* at costovertebral angle.

Urine

Hazy or cloudy. Loaded with pus cells, some red blood cells. Sterile to routine culture Stained sediment reveals typical bacillus.

Cystoscopic data

Bladder capacity limited, bladder markedly irritable. Mucosa markedly congested, with tubercles *in neighborhood of* ureteral orifice on affected side. Later, ulcerations are observed Extensive involvement of entire bladder follows

Meatoscopy

Urinary efflux difficult to see. Area surrounding ureteral orifice shows hyperemia, tubercle formation and later ulcerations. Ureteral orifice is distorted, may be elevated or crater-like (golf hole) Entire area surrounding orifice appears rigid and stiff

Ureteral catheterization

Ureter may be readily catheterized or be occluded, preventing catheterization to kidney Urine, when collected, pale but cloudy, low specific gravity, acid. Sediment reveals pus and tubercle bacilli.

Function tests

Phenolsulfonphthalein

If ureter is occluded, no specimen, or delayed, impaired percentage of functional ability.

Indigo-carmine

Delayed function or faint or no concentration.

X-ray

Plain May reveal old calcified lesion.
Pyelography · Shaggy, moth-eaten area of the calyx—major or minor calices

Symptoms

kidney
Symptoms referred to
Not a prominent symp-
Pain
tom Usually no pain until late in the disease.

Constitutional symptoms

Loss of weight and energy. As the disease advances, chills, night sweats, progressive emaciation.

Bladder symptoms

Prominent, may be only symptomatology. Marked frequency of urination, smarting and burning on urination, Occasional hematuria Irritable pyuria and polyuria.

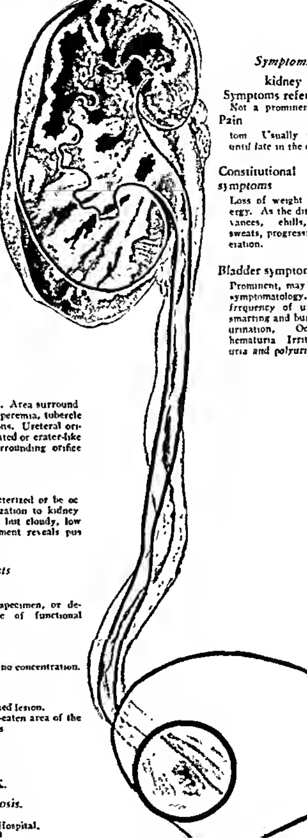


PLATE LXX.

Renal Tuberculosis.

(Temple University Hospital, Acc. No. 163.)

is by far the most important. Renal tuberculosis may occur in either sex, at any age; it may be unilateral or bilateral. It has been definitely established that renal tuberculosis is a local manifestation of a constitutional disease. It has also been confirmed that there is a definite predilection of the tuberculous infection to spread to the organs of the associated genital system; the prostate, seminal vesicles and epididymi. The opposite is likewise true that involvement of the epididymi, prostate, or seminal vesicles is followed by tuberculous lesions of the kidney. It is believed that so far as the urogenital system is concerned, the kidney is the most frequent initial site of the infection and that the epididymi, prostate, and seminal vesicles are secondarily invaded. Bumpus, in a survey of 606 cases of genital tuberculosis, found kidney involvement 330 times. It may be impossible, in the presence of a combination of lesions, to determine the site of the initial lesion of the urogenital system. Once a diagnosis of urogenital tuberculosis has been made, a careful and systematic examination of the entire genitourinary system is indicated. The author has observed two men, 19 and 27 years of age, who submitted to orchidectomy because of a unilateral ulcerative tuberculous epididymo-orchitis. Both were without urinary symptoms. Examination revealed the presence of a progressive tuberculous process of one kidney. Both men submitted to nephrectomy and were ultimately cured. A highly important fact was that at no time did either complain of urinary symptoms and only by routine examination was renal tuberculosis discovered.

Ordway and Medlar, in a ten years' survey, found that among the known tuberculous patients, 7.7 per cent showed tubercle bacilli in the urine. Of these, 77 per cent had no clinical symptoms suggestive of renal tuberculosis.

Age and Incidence: Renal tuberculosis is predominantly a dis-

ease of early adult life. Caulk found the average age to be 31½ years. G. J. Thomas, in a survey of 153 cases, found the average age was 32 years; the youngest was two years of age; the oldest was 81. Wildbolz found the occurrence of renal tuberculosis between 20 and 40 years in 71 per cent of 315 cases. These figures are those in which renal tuberculosis was known to exist. Statistical data of the incidence of renal tuberculosis in general surveys show more clearly the frequency with which renal tuberculosis occurs. Selleras and von der Becke, in an analysis of 1724 individuals with urinary symptoms, found the occurrence of renal tuberculosis in 16 per cent. Kuester, in 5338 autopsies, found 158 cases or relatively 3 per cent. Kapsammer, in an analysis of 20,777 autopsies, found renal tuberculosis in one per cent of the cases. G. J. Thomas found its occurrence in 17,777 autopsies to be 0.62 per cent.

Pathology: Renal tuberculosis is a hematogenous or blood-borne infection. It results from the introduction, implantation and germination of the tubercle bacillus in the kidney from a primary focus elsewhere in the body. The primary focus is usually the lungs. The infection spreads by way of the lymphatics to the mediastinal lymph nodes. The tubercle bacillus may be swallowed (bovine bacilli in milk), and lodge in the intestinal mucosa with the formation of a tubercle. The bacteria are picked up by the lymph channels and carried to the mesenteric lymph nodes. The primary lesion in the lung or intestine may heal or progress. The metastatic implantation in the lymph nodes may remain quiescent for years, to become active long after the primary lesion has healed. Invasion of the blood stream ultimately occurs by way of the lymphatic system through the thoracic duct. This tuberculous septicemia or bacillemia may be overwhelming, with the production of a generalized miliary tuberculosis. The infective emboli may be carried to the bones, joints, kidneys or other distant organs or areas

with the production of definite and typical tuberculous lesions at the site of lodging

It is possible that renal tuberculosis may result from a secondary lesion elsewhere in the body (bone, joint, genital organ), or that both the renal lesion and the other secondary lesions may have their origin from the same primary focus in the lungs or intestines

It is now quite generally recognized that renal tuberculosis is a secondary hematogenous infection and is never primary in the kidney It has been believed by some observers that the presence of tubercle bacilli in the urine does not signify or prove the occurrence of renal tuberculosis Cohnheim first advanced the theory that such organs as the liver, intestines, and kidney can excrete bacteria from the blood stream as a sort of physiologic function Wyssokowitsch, in 1886, refuted this theory, but many still believe the fallacy of the Cohnheim theory as an easy explanation for bacilluria In recent years, Helmholz, Montgomery, and Allen, Liberthal and von Huth, have conclusively shown that the normal kidney cannot physiologically excrete the tubercle bacillus and that the finding of the tubercle bacillus in the urine, withdrawn directly from the kidney, proves conclusively the presence of renal tuberculuosis

Medlar believes that renal tuberculosis is bilateral during the stage of invasion He has clearly demonstrated this fact in experimental work with animals and in the examination of microscopic sections of kidneys obtained from humans who have died of tuberculosis Medlar advocates examination of serial sections of the whole of both kidneys in proving the possibility of minute healed processes This work has been substantiated by Band who demonstrated bilateral tuberculous lesions in each of five individuals who, during life, excreted tubercle bacilli, but at autopsy no macroscopic evidence of renal tuberculosis could be found Hin

man, however, refutes this by stating . . . "The assumption of initial bilaterality is unwarranted. There is no reason to assume the infection of both kidneys, because the infection is blood-borne, than to assume that both knees must be infected because one is, or both femurs, or any other two similar parts of the body. Furthermore, staphylococcal renal infections, which are always hematogenous, are usually unilateral, even when acute and fulminating."

There is little evidence clinically that a single kidney is primarily infected by the tubercle bacillus and that its fellow is subsequently infected by an ascending infection from the bladder by way of the ureter. Thomas and his associates believe that bilaterality occurs in 60 per cent of cases during the stage of invasion and the incidence of bilateral infection will be greater if earlier and more careful examinations of the kidney are made. In their experience, bilateral infection occurs in 46 to 51 per cent of early and late lesions of renal tuberculosis.

Undoubtedly a great degree of the misunderstanding of such a vital subject rests with the interpretation of the pathologist. Beyond question, the microscopic scars of healed tuberculous lesions of the kidney do not materially differ from those produced by other chronic inflammations. These minute scars are generally overlooked or are not considered as healed tuberculous lesions.

This highly controversial point regarding the spontaneous healing of early tuberculosis is still debated by some investigators. The investigative work of Medlar, Band, Thomas, Stebbins, and Sandell; Beach and Shultz; Baggenstoss and Greene; Keyes, Putschar, and others have shown conclusively that the early lesions of tuberculosis do heal spontaneously in some instances. It can then be assumed that the simultaneous hematogenous implantation of tubercle bacilli in both kidneys will heal in some instances. In

other instances, the healing process may be on one side only, while on the other side, destruction of kidney tissue continues until the diseased kidney is removed either on the operating table or on the autopsy table. By the same token, it is a generally conceded fact that the presence of tubercle bacilli in the kidney urine proves a tuberculous disease in a given kidney. The sustained disappearance of the organisms from the kidney urine, with no return for years as shown by repeated laboratory tests, should attest clinical healing of the former lesions.

It is generally assumed that renal tuberculosis follows the lodging of a bacillus-laden embolus. The site at which such an embolus lodges has been a subject of much conjecture. Medlar, following extensive study of the kidneys of tuberculous patients who did not have urinary symptoms, and in experimental tuberculosis in animals, came to the conclusion that there were three types of renal lesions: 75 per cent were cortical; 11 per cent were medullary and 13 per cent were cortico-medullary. The cortical lesions are believed by some writers to be usually a part of miliary renal tuberculosis and are nonprogressive. The medullary lesion usually grows relatively fast. Medlar believes that such lesions occasionally heal, but more frequently the tubercle will spread, opening ultimately into a renal tubule or papilla and then be able to discharge its contents into the renal pelvis. If the medullary tubercle is near a calyx, the clinical symptoms of renal tuberculosis will appear relatively early; if at the base of a pyramid, a considerable portion of the kidney parenchyma will be involved before pelvic infection occurs. Both Lieberthal and Wildbolz believe this type of renal tuberculosis develops first in a renal papilla. Medlar, after his extensive and exhaustive study, concluded that the smaller lesions were of two types. One of vascular origin within the capillary tuft of a glomerulus, or within a capillary be-

tween the convoluted tubules in the pyramids. The commonest was the lesion within a glomerulus. The second type of lesion was found to have its origin within the lumen of a tubule either in the lowest point of the loop of Henle or in the collecting tubules in the pyramids. This type of lesion was always secondary to an ulcerating lesion higher up in a tubule and tubercle bacilli were discharged into the lumen of the tubule.

The lesions of renal tuberculosis may be grouped:

1. Acute bilateral miliary tuberculosis.
2. Chronic renal tuberculosis.

1. Acute bilateral miliary tuberculosis of the kidney usually occurs as part of a generalized miliary tuberculosis. As the clinical picture is one of rapid, fatal termination, there is little that can be done so far as treatment is concerned, except to maintain comfort for the patient. The general condition is so rapid that sufficient time does not elapse to permit caseation and cavity formation in the kidney. The kidney, as well as other structures, is studded with tubercles one to two millimeters in diameter. The urine contains tubercle bacilli but no pus cells. The condition is generally one of infancy and childhood. Mathé has pointed out that the younger the child, the more rapid the progress of the disease. Caulk believed that in the less virulent cases, healing could take place if resistance to the generalized bacterial dissemination could be maintained.

2. Chronic renal tuberculosis is the form which is of the most *importance to the urologic surgeon (Fig. 329). Chronic renal* tuberculosis is, within itself, a progressive, destructive lesion. Usually, the initial lesion begins as a typical tubercle in or near the immediate neighborhood of a pyramid. The tubercles tend to spread and coalesce (Fig. 330), always showing a tendency to progress. Tuberculous lesions, once established, vary so much in appearance that no single description could apply to them all.

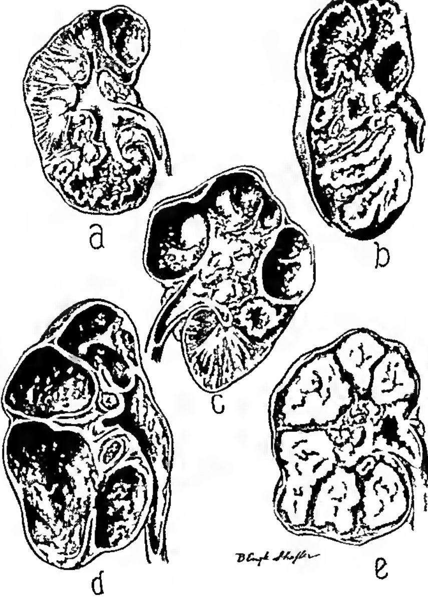


Fig. 329—Renal tuberculosis

They vary from a simple nodule within a pyramid; a simple ulceration of a papilla; cavity formation involving one pole of the organ (FIG. 331), to complete destruction of the kidney (FIG. 332), as the result of either pyonephrosis or caseation with fibrosis and atrophy. In many instances, the kidney will show several different types of lesions when examined after removal (FIG. 333).

The variegated appearance of the lesions depends greatly upon the activity and virulence of the organism, the resistance of the individual to the invading organism and the condition of the ureter. Early involvement of the ureter, with resulting urinary stasis, does much to intensify the destructive process.

A. Ulcerative cavernous type, moderately advanced. Small cavity in apex not communicating with pelvis. Lower calyx destroyed. A single, deep-lying focus embedded in the parenchyma involving one of the pyramids but having no communication with the pelvis. Ureter shows no anatomical change.
Retrograde pyelography would show erosion in lower portion of kidney.
Intravenous urography—some delayed function with evident erosion of lower calyx.

B. Ulcerative cavernous type, advanced. Large cavity involving upper portion. Middle portion shows erosion of the parenchyma without communication with the pelvis. Lower portion shows extensive ulceration of parenchyma with communication with the pelvis. Ureter thickened but patulous.
Retrograde pyelography—cavitation and ulceration.
Intravenous urography—delayed function of poor concentration.

C. Mixed type. Advanced stage. Marked cavitation of upper portion having communication with pelvis. Middle portion shows an ulceration with communication with the pelvis. Lower pole functionally normal. Ureter thickened but patulous.
Retrograde pyelography—cavitation of upper portion of kidney.
Intravenous urography—poor, delayed function.

D. Complete destruction of kidney. Entire kidney parenchyma converted into large, irregular cavities. Ureter thickened, partially or completely occluded.
Retrograde pyelography—if ureter is open, marked multiple cavitation involving entire kidney. If ureter is closed, pyelography impossible.
Intravenous urography—functionless kidney.

E. Complete destruction of kidney. Masses of caseous material occupy the areas between the columns of Bertim. Marked deposition of lime salts is present which will be seen on the roentgenogram. The ureter is dilated, thickened and closed.
Retrograde pyelography—impossible.
Intravenous urography—functionless kidney.

There are three principal groups of pathologic changes produced by the invasion of the tubercle bacillus:

(*a*) The caseocavernous type.

(*b*) The disseminated nodular type.

(*c*) The fibrous or indurative type.

Fig. 330—Renal tuberculosis. Multiple caseous areas of tuberculosis involving widely separated areas of the kidney
(Temple University Hospital, Acc No. 3316)

(*a*) *The caseocavernous type* of chronic renal tuberculosis is the most common form If the original medullary lesion is in the form of a minute ulceration on the surface of the papillae, with early infiltration of the neighboring calyx, clinical signs will be quickly manifest However, if the lesion originates deep in the parenchyma at the base of a pyramid, it may attain a considerable

size before reaching the pelvis and the establishment of symptoms. Once established, these lesions show little tendency to heal but continue to progress to a more advanced stage. The fully advanced stage is marked by characteristic changes in color, contour and consistency of the kidney. The end stage shows marked lobu-

Fig. 331—Renal tuberculosis. Localized caseous tuberculosis of the lower pole of the kidney.
(Temple University Hospital, Acc. No. 3635.)

lation and, finally, renal destruction. Of all the forms of chronic renal tuberculosis, the caseocavernous type is the commonest. The lesions in the advanced stages may be most marked in the parenchyma or the lesion may involve principally the pelvis and ureter.

Calcification frequently occurs in association with caseation. The entire kidney may be calcified or there may be a single dense

Fig. 332—Renal tuberculosis Complete destruction of the parenchyma of the kidney Nothing remains except the thin outer fibrous capsule
(Temple University Hospital Acc No 7889)

area of calcification which in the roentgenogram, may appear as a single calculus The calcification is not usually of uniform consistency but presents a moth eaten appearance More frequently small calcareous deposits appearing singly or in groups, may be seen irregularly scattered throughout the roentgenogram of the kidney The tubercle bacillus frequently invades a kidney already subjected to diseased conditions such as calculus hydronephrosis tumor polycystic disease complicating the clinical picture of both conditions Perinephritis is relatively common in its occurrence in renal tuberculosis Perinephritic abscess is not an uncommon occurrence

Bugbee recently reported a true tuberculoma of the kidney in

Fig. 333—Renal tuberculosis. Widespread caseous tuberculosis. (Temple University Hospital, Acc No 163.)

which the tuberculous process presented a definite tumor formation. There was neither necrosis nor cavity formation within the nodule although active *tuberculosis* and *rapid formation of tubercles* was found throughout the kidney (Figs. 334, 335).

(*b*) *The disseminated nodular type:* This is a rare type of renal tuberculosis. The renal parenchyma is replaced by a diffuse corticomedullar fibrosis and numerous areas of conglomerate tubercles. Cavity formation is absent.

(*c*) *The fibrous or indurative type:* The outstanding feature of this sclerotic form is the widespread fibrous tissue change and the almost complete lack of tubercles. The tubercles are so few in number that prolonged microscopic search may be necessary to

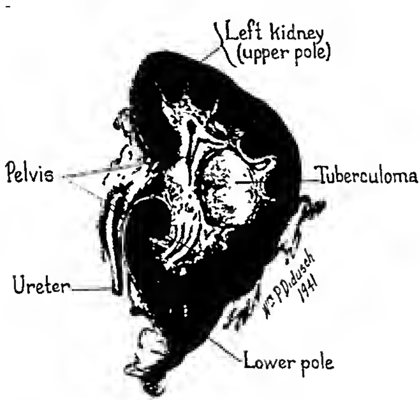


Fig 334—Tuberculoma of kidney Tuberculoma fills the renal pelvis
(Courtesy of Dr Henry G Bugbee and *Journal of Urology*, 1941, 46 355)

locate a single one. The fibrous tissue changes may be so extensive as to cause a marked reduction in the size of the kidney, and on microscopic examination, a histologic picture not unlike that of a secondary contracted kidney of chronic glomerulonephritis may be seen.

Ureteral and Vesical Lesions: Tuberculous involvement of the

ureter and bladder appears quite early in some instances; late in others, or may never occur. *It is usually the rule that involvement* of these structures does occur. Once the medullary tubercle has ruptured into the renal pelvis, tubercles appear on the mucosa of

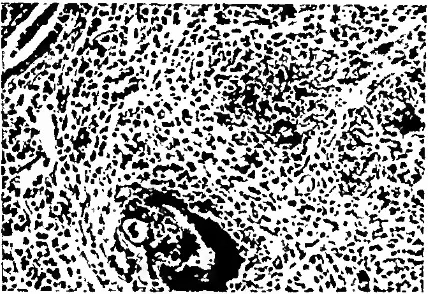


Fig. 335—Tuberculoma of kidney. Microscopic section shows endothelial cells, lymphoid infiltration and giant cells.
(Courtesy of Dr. Henry G. Bugbee and *Journal of Urology*, 1941, 46,355.)

the renal pelvis. The tubercle bacilli are carried down by the urine, implanting themselves in the ureteral wall, producing characteristic lesions. The lesions are usually near the lower end of the ureter although the condition may be diffuse. In some instances, the tubercles break down forming distinct ulcerations on which calcareous deposits may take place. Again, the ureter may show definite stricture formation as the result of the infection invading the deep coats of the ureter. The ureter may show extensive calcification throughout its entire length. Dilatation of the ureter is common as the result of the inflammation and partial occlusion. When total occlusion of the ureter occurs, either as the result of

calcification or by caseous material autonephrectomy results The final result of autonephrectomy is a pyonephrosis a complete calcification or an atrophied kidney The occluded kidney is a definite menace because of the constant danger of a generalized miliary tuberculosis resulting from the virulent bacilli within the caseous filled cavities

As the infected urine is continuously passed into the bladder, the first vesical changes are usually around the corresponding ureteral orifice A greater area of the bladder gradually becomes involved Later ulcerations occur the musculature becomes infiltrated and subsequent contraction occurs There is rarely caseous degeneration of the bladder as is seen in the kidney

Symptoms It is an established fact that the early lesions of renal tuberculosis exist both clinically and pathologically without producing symptoms It is likewise true that renal tuberculosis may be fulminating producing very severe initial symptoms The disease usually develops so slowly and insidiously that it may be rather far advanced before patients come under observation The majority of patients are well nourished and apparently in good health during the early stages of the disease

As the result of the continuous irritating bladder symptoms loss of sleep loss of appetite, and the fear of drinking fluids these patients suffer with fatigue and lowered resistance with consequent impairment of their general physical condition They may fall victims to the ravages of an intercurrent infection

Patients are usually made worse by local bladder treatments Tuberculosis of the bladder and kidney should always be suspected in the presence of a chronic cystitis that is not amenable to ordinary methods of treatment

1 *Urinary disturbances* Frequency of urination is the most common symptom It is the first symptom in 75 to 85 per cent of

the cases. The onset is gradual, but on direct questioning it will be learned that the time between urination will be decreased. As the disease progresses, the frequency may be so increased, during both the day and night, that only a few moments elapse between voiding. Sleep or rest is impossible due to the almost constant desire to empty the bladder. Frequency of urination is usually due to tuberculous ulceration of the bladder.

2. *Urgency of urination:* At the onset of involvement of the bladder, the desire to empty the bladder may be controlled. As the frequency increases in intensity, it is observed that there is also an inability to control the emptying of the bladder. Urgency may become so acute that once the desire to void arises, the urine must be passed immediately or incontinence ensues.

3. *Pain on urination* is usually a relatively late symptom. As the infection spreads to the vicinity of the bladder neck, pain may be marked just before, during or after urination. The irritation at the vesical neck may be so acute that a reflex spasm of the vesical sphincter is produced. This reflex spasm is known as tenesmus or strangury. Due to the marked inflammatory reaction present, expulsion of a few drops of blood may occur at the end of each urination (terminal hematuria).

4. *Bladder capacity:* As the symptoms referable to the bladder develop and increase, the capacity of the bladder may be reduced to 50 or 100 cc. As the infection spreads into the muscular coats of the bladder, a condition known as "contracted bladder" may develop, so that urination is necessary during both the day and night, at very frequent intervals.

5. *Pyuria* becomes apparent with the first ulceration of the renal papillae and is continuous throughout the course of the disease. Closed parenchymal lesions may become extensive with almost complete destruction of one pole of the kidney without communicating with the renal pelvis. The extensive nature of the

lesion cannot be estimated by the extent or degree of pyuria present The presence of renal tuberculosis should always be suspected in the presence of pyuria in an acid urine that is negative to ordinary culture

6 *Hematuria* The occurrence of blood in the urine is an important first symptom although it occurs in only five per cent of all cases Hematuria may be renal or vesical in origin Its presence is more frequently observed in bladder involvement and may be noted at the end of each urination The passage of long thin clots accompanied by renal pain may be such that renal calculus is suspected Periodic hemorrhages may occur, the urine being clear between such attacks Such bleeding is suggestive of a relatively mild tuberculous process rather than a severe fulminating infection Usually, copious bleeding occurs only when the ulcerations are well advanced

7 *Pain* There is nothing characteristic or pathognomonic of such a symptom There may be a constant, dull, aching pain or a feeling of heaviness in the lumbar region of the affected side The pain may occasionally be acute, simulating a renal colic and is due to the passage of a blood clot or caseous material through the ureter

Pain over the suprapubic area is usually of a smarting, burning character and is associated with extensive tuberculous lesions of the bladder

8 *Genital involvement* Many patients are observed whose first and only symptom is a subacute or a recurrent unilateral epididymitis Urogenital tuberculosis should be suspected in any case of epididymitis in which gonorrheal urethritis and prostatitis can be excluded Repeated examinations of the kidneys and kidney urine may be necessary to establish a diagnosis of renal tuberculosis It is believed that genital tuberculous lesions are always secondary to renal tuberculosis

Diagnosis: There are several suggestive features that should always be considered and search made for tuberculosis.

A young individual of either sex, presenting a history of frequency and urgency of urination, noctnria and polyuria, and on voiding, the urine is found to contain pus cells; or a patient exhibiting protracted and recurrent cystitis without demonstrable reason; or a male exhibiting recurrent epididymitis where gonorrheal urethritis and prostatitis can be excluded, should be examined carefully for tuberculosis.

History: A carefully elicited history may be of the utmost importance yet it may be entirely negative. A history of a preëxisting tuberculosis may be of the greatest importance, but often such a history may be entirely lacking. Renal tuberculosis is definitely a local manifestation of a constitutional condition and by the time the renal lesion is observed by the urologist, the local condition may be rather far advanced. A patient between the ages of 20 and 40, who presents a protracted history of frequency, urgency, nocturia and pain on urination, is presenting very pertinent data that should be carefully analyzed and the patient subjected to complete urologic examination to the exclusion of renal tuberculosis.

Examination: Palpation of the kidney is of little aid in the early phases of the disease. It is not until the disease is well advanced that the kidney is sufficiently enlarged to be palpated. In males, a careful examination of the genital tract should be made: palpation of the epididymes may disclose an enlarged, firm, nodular epididymis. Rectal examination may reveal nodules in the prostate that are firm and sharply demarcated.

Urine examination: Rontine urinalysis will reveal the presence of a highly acid urine, pus, blood, albumin and possibly a few casts. A sterile culture of highly acid pyuric urine is presumptive but not conclusive evidence of tuberculosis. The tubercle bacilli

must be demonstrated and isolated before a diagnosis of renal tuberculosis can be made It is an established fact that a tuberculous kidney will intermittently discharge tubercle bacilli into the urine and that the urine will be negative one day and positive the next For this reason it is imperative to make repeated tests at regular intervals Examinations should be made of 24 hour specimens

There are four methods of examination of the urine for the demonstration of the tubercle bacilli

1 Staining the sediment of a 24 hour specimen by an acid fast technic

2 Fluorescent microscopy Auramin stain

3 Guinea pig inoculation with a portion of the sediment of a 24 hour specimen The animal is sacrificed after six weeks G J Thomas believes that The guinea pig is the most trustworthy laboratory animal that constantly furnishes an indisputable test of the presence of bacilli of tuberculosis in the urine We believe that the guinea pig will reveal evidence of tuberculosis if a sufficient number of bacilli of tuberculosis are injected into the animal s peritoneal cavity

4 Cultural identification of the tubercle bacillus in the urine It is believed that a cross check by cultural methods is very advantageous Animals following inoculation with urinary sediment often die of peritonitis Considerable difficulty and inconvenience to the patient may be averted by having instituted cultural methods at the time of animal inoculation The value of identification is relatively equal between cultural methods and animal inoculation The culture medium used is the Petragnani medium

Intravenous urography is an ideal method to visualize the kidneys and to ascertain their functional ability It is believed that retrograde pyelography is the better means to determine the destructive lesions of renal tuberculosis Intravenous urograms are

seldom sufficient to outline the minute filling defects of the ulcerative lesions. In certain instances where the ureteral orifices cannot be visualized or some obstruction prevents the passage of ureteral catheters, intravenous urography must be used alone. It should be routinely used before cystoscopy is done because considerable information may be gained: the presence or absence of anomalies; the position of the kidneys; the relative functional ability of both kidneys; the presence or absence of gross destructive lesions. All these will aid materially in the complete summary of the individual case under study.

Cystoscopy: When examination of the urine and other clinical evidence suggests or proves the presence of genitourinary tuberculosis, a complete urological examination should be done. Only by this means can an accurate diagnosis of renal tuberculosis be established. Cystoscopy with ureteral catheterization, collection of ureteral specimens, functional kidney tests and pyelography should be done in every instance. It is only through the meticulous interpretation of the findings of such an examination that any evaluation of the extent of the condition or the extent or method of treatment can be intelligently outlined.

Anesthesia for cystoscopic examination: Anesthesia is usually necessary due to the increased irritability of the bladder in vesical involvement by tuberculosis.

After the genitalia have been cleansed, a local surface anesthesia may be used in both male and female. In the female, 10 to 15 cc. of one per cent diothane solution is injected into the urethra by a sterile urethral syringe, following which a cotton applicator is inserted within the lips of the urethra, having first been saturated with a ten per cent cocaine solution. In the male, 10 to 15 cc. of a one per cent solution of diothane is injected through the urethra A cotton applicator, saturated with a ten per cent cocaine solution.

is inserted into the meatus. This technic is usually sufficient to permit routine manipulation.

It is imperative, in using diothane solution, to wait a minimum of five minutes before introducing the cystoscope. The injection of 30 cc. of one per cent diothane solution, or a similar amount of two per cent metycaine solution, may render an irritable bladder sufficiently anesthetic to permit rather extensive manipulation.

In the presence of severe bladder symptoms, it may be necessary to use spinal anesthesia, 50 to 60 mg. of novocain injected at the level of the fourth lumbar interspace into the subdural space, will render sufficient relaxation and nerve block to continue cystoscopy without pain or trauma. Spinal anesthesia is to be preferred to inhalation anesthesia although there is no contraindication to the latter unless there is a pulmonary lesion. Intravenous anesthesia may be used with good results. It is believed by the author that of all the anesthetics at one's command, local anesthesia is to be preferred, providing extensive ulceration and contraction of the bladder are not present.

As has been previously stressed, introduction of the cystoscope and cystoscopic manipulation should be done only with the greatest of skill and gentleness. This dictum is again repeated because in the presence of vesical tuberculosis, trauma will add materially to the already great discomfort of the patient.

Before the cystoscope is introduced, every portion of the sheath that touches the mucosa of the urethra should be thoroughly lubricated. After introduction of the cystoscope into the bladder, the obturator is withdrawn and a specimen of bladder urine is collected directly into a sterile test tube. The bladder cavity is then irrigated with sterile water until the return flow is clear. A complete systematic examination of the entire bladder mucosa and ureteral orifices is then made.

Cystoscopic appearance of the bladder: The cystoscopic ap-

pearance of the bladder mucosa in the presence of tuberculosis may vary according to the stage of the process. The lesions regarded so characteristic are the tubercles and the tuberculous ulcerations. It should be remembered that the tubercles are present at only one stage and are not always present when the patient is examined. Vesical tuberculosis in the early stage of the disease presents itself as a limited inflammatory areola in relation to one ureteral orifice. Tubercles then appear but disappear in the later stages when an extensive sloughing ulcerative cystitis is present.

Hyperemia: The earliest lesion of vesical tuberculosis is a triangular area of redness lying on the trigone below the ureteral orifice on the affected side. There are no tubercles present. Such hyperemia is not pathognomonic but may result from a renal infection. There is gradual infiltration of the ureteral orifice as the ureteral walls are invaded by tubercles.

Tubercles: As the ureteral walls are invaded by tubercles, the vesical mucosa near the ureteral orifice on the affected side is also invaded. The bladder mucosa near the orifice is usually involved rather than the trigone. The tubercles appear as small, discrete, yellowish elevations with a distinct limited areola which quickly fades away into normal, healthy appearing mucosa. Single tubercles are uncommon. The tubercles usually occur in small groups. The groups are never widely spread over the bladder surface but are confined to rather circumscribed areas. The tubercles are usually in close apposition to the blood vessels and may be occasionally seen lying along the course of a branching artery. The tubercles tend to remain localized in a limited area, but sooner or later they break down with the formation of ulcers.

Bullous edema: Bullous edema formation may be extensive or limited to a cluster surrounding the ureteral orifice of the affected side, although it is not a pathognomonic or characteristic factor.

Ulceration: Tuberculous ulcers are at first shallow, irregular ulcers with undermined edges. The base is usually covered with a dirty-yellowish-gray slough, which may be of sufficient size to hide the ulcer completely. Surrounding the ulceration at irregular points may be seen tubercles which will soon rupture, extending over the area of the ulceration. At first the ulcerations are shallow, but they show definite tendency to progress, to become deeper and to spread over wider areas. They may coalesce with other ulcers. The sites of these ulcers are not only in the vicinity of the ureteral orifice but also in the dome of the bladder. The latter is believed to be the result of contact when the bladder is empty. In the later stages, the effect on the trigone may be startling. The edges of the trigone may be so undermined by the ulceration that the trigone is held to the bladder only at the corner attachments. Such a case has been reported by Young.

The ureteral orifice displays quite characteristically the changes that are taking place in the bladder. In the first stages, when hyperemia was the only visible evidence of renal tuberculosis, the ureteral orifice becomes infiltrated, rigid and appears elevated. It also loses its mobility, remains fixed and appears to be uninfluenced by the normal peristaltic wave. The orifice may subsequently be completely obscured by bullous edema As the inflammatory process in the bladder continues, the ureter becomes infiltrated and rigid and foreshortened with the production of the typical "golf-hole" ureter, in which the orifice becomes and remains patulous and stands out discretely as an open, retracted hole. The ureteral orifices may be obscured by extensive ulceration and contraction of the bladder. If visible, both ureteral orifices may present similar appearances, making it impossible to differentiate which side was originally affected. As contraction of the bladder continues, the ureteral orifices appear to lie high on

the posterior wall of the bladder due to the fact that the trigone is not contracted in the same ratio as the remainder of the bladder.

Collection of ureteral specimens: The ureteral catheter is inserted into the catheter guide of the cystoscope. To the distal end of each catheter is attached, by means of a needle or adapter, a 30 cc. Luer syringe filled with sterile water. In this way, the catheters are continuously flushed by a slow, steady stream of sterile water while passing through the cystoscope, bladder and the ureteral orifices. Injection of the water is stopped immediately after the eye of the catheter is advanced past the ureteral orifices. The syringe is detached. This continuous flushing of the catheter is done to prevent contamination of the lumen of the catheter by the infected urine of the bladder. The catheters are advanced up the ureters and specimens from each kidney are taken (10 to 20 cc.). Intravenous injection of indigo-carmine or phenolsulfonphthalein may then be done for individual renal function tests.

Pyelography: Following the function tests, pyelography is done on one side only, the opposite side to be done at a later examination. Skiodan, 20 per cent solution, is routinely used as the radiopaque solution. The patient is placed in a modified Trendelenburg position, the catheter on the selected side is attached to a burette by an adapter. The skiodan solution is permitted to flow slowly into the renal pelvis by gravity until a sensation of fullness, but never of pain, is experienced by the patient. The x-ray exposure is made without moving the patient. A stereoscopic pair of films is always exposed. These films are immediately developed and examined in the dark-room. If filling of the pelves and calices is complete, the catheters and cystoscope are withdrawn. If the filling is incomplete, the pelves and calices are refilled with the opaque solution and another stereoscopic pyelogram made. The patient is again cystoscoped and the opposite kidney injected and pyelogram made after four or five days. By observing rigid

cystoscopic technic there is little or no danger of engrafting an infection on the uninfected kidney

G J Thomas and his associates do not believe that tuberculosis

Fig 336—Retrograde pyelogram. Renal tuberculosis with extensive involvement of the ureter The calyces are distorted and show evidence of ulceration.
(Philadelphia General Hospital)

is spread to the uninfected side by pyelography stating we have never observed a patient in whom the making of a retrograde pyelogram was even a remote factor in producing the spread of tuberculosis from one area in the kidney to another or into the blood stream

Roentgenological appearance The filling defects of destructive lesions of renal tuberculosis may be classified

1. An ulcerative lesion produces a shaggy, irregular, "moth-eaten" appearance on one of the minor calices (Fig. 336).

2. A parenchymal abscess appears on the pyelogram as a filling

Fig. 337—Retrograde pyelogram. Renal tuberculosis. Note widely separated collections of the contrast medium.
(Philadelphia General Hospital)

defect by appearing as an accessory pocket arising from a normal calyx or pelvic shadow. There may or may not be a demonstrable connection. These abscesses vary from very small to large excavations (Fig. 337).

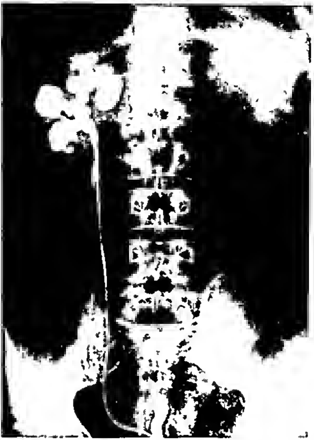


Fig 338—Retrograde pyelogram Renal tuberculosis. Note lobulated appearance Areas of ulceration may also be seen. There is no apparent involvement *of the ureter except at the upper extremity*
(Temple University Hospital, Acc No 48444)

3. Pyonephrosis presents a typical loculated appearance that varies little from a similar condition produced by other causes. The normal pelvic outline and renal parenchyma may be so destroyed that only a pyonephrotic sac remains (Fig. 338).

Urine studies: The three (bladder, right and left ureteral urine) specimens collected at cystoscopy are subjected to the following studies:

1 Routine culture for secondary organisms; aerobic and anaerobic culture.

2. Smears of centrifuged sediment for acid-fast organisms.
3. Fluorescence microscopy—Auramin stain.
4. Guinea-pig inoculation of the sediment from bladder, right and left ureters.

Prognosis: It is believed that the prognosis of renal tuberculosis is more favorable in the instances of unilateral tuberculosis that have been subjected to nephrectomy. It is best to remove a kidney so progressively destroyed before serious involvement of the bladder occurs. It is surprising how great an improvement of the bladder will follow the removal of the infected kidney. Statistics uniformly agree that the period of longevity is greatly increased by surgical removal of such a diseased kidney, as compared with strictly medical care.

Bilateral renal tuberculosis may be benefited by surgery on one side, or it may be necessary to restrict treatment to strictly medical and hygienic principles. Such decision regarding treatment can only be determined by the interpretation of the numerous studies of each individual case.

Treatment: The treatment of renal tuberculosis is not strictly medical and not strictly surgical, but a combination of the two. The renal lesion is a local manifestation which, in itself, usually requires surgery. The surgeon should coöperate with the physician in the management of the tuberculous disease. Nephrectomy should be done only if the lesion in the kidney exhibits a definite progressive, destructive tendency and the opposite kidney is normal and healthy. Never should nephrectomy be done just because the tubercle bacilli are found in the urine. If bilateral renal involvement is found and is equally advanced, conservative treatment only should be considered. If one kidney is completely destroyed and the other kidney shows a nondestructive lesion and possesses good function, then nephrectomy is warranted. In bilateral involvement of the kidneys, surgical treatment should not be

done if the lesion is small on both sides or small on one side and moderate on the other The duration of life of individuals having bilateral renal tuberculosis depends upon the amount of normally functioning renal tissue It is imperative to maintain all possible reserve

BILHARZIASIS OF THE KIDNEY

Infection of the kidney and upper urinary tract with *Schisto soma haematobium* is rare although involvement of the bladder and lower ureter is common in those countries where the organism is prevalent Pijper in discussing the pathology of the disease states that the kidneys ureters and urethra are hardly ever in fected Vermooten has definitely demonstrated that the lower ureters are frequently involved while the kidneys remain normal A R Stevens reported a case in which the bladder and ureters were involved but there were papillomatous lesions also involving the renal pelves and calices Complete nephroureterectomy in two stages was followed by complete recovery

Symptoms of renal bilharziasis are not pathognomonic but are those of pain in the lumbar region and palpable tumor mass on the affected side Urine examination may show the presence of albu min a few red blood cells and the characteristic ova

Cystoscopy Unless there are characteristic lesions within the bladder the appearance of the bladder will not be suggestive of upper urinary tract involvement Owing to the nature of the offending parasite the bladder will usually show typical lesions

XXV

SPECIFIC INFECTIONS OF THE KIDNEY

Continued

SYPHILIS OF THE KIDNEY

Nephritis is considered to be a rare manifestation of syphilis. Although seldom mentioned in our textbooks, syphilis of the kidney may and does occur at any stage of the disease. Beyond question, many instances of syphilitic nephritis have occurred without ever being recognized or having been treated as such.

Pathology: The acute nephritis occurring in the early stage of syphilis or during the first year after initial infection, presents a highly characteristic clinical picture. Some investigators state that the picture is so striking and characteristic that if routine biochemical tests are applied, the nature of the nephritis will be demonstrated even though there is an absence of other signs of syphilis.

In the later periods, that is, the latent period following the secondary manifestations and in the tertiary stage of syphilis, a characteristic clinical picture is not presented. The symptoms and the urinary findings are usually such that there is no clue that the nephritis is of syphilitic origin. Fournier believed that the pathological anatomy of chronic nephritis resulting from syphilis bears no specific significance. The occurrence of gumma is rare (FIG. 339) and even though a gumma may be recognized as such, the sclerotic changes occurring are indistinguishable from interstitial changes resulting from other causes (FIG. 340).

Fournier described two principal types of chronic syphilitic nephritis:

1. Chronic diffuse parenchymatous nephritis, which grossly appears as a large, white kidney. Microscopically, there is granular and fatty epithelial degeneration, which is most marked in the

Fig 339—Gumma of the kidney.
(Courtesy of Dr Archibald W. Hunter and *Journal of Urology* 1939, 42 1176)

convoluted tubules, proliferation of the epithelial lining of Bowman's *capsule and cellular infiltration* of the *interstitial tissue*. Progressively marked glomerular and interstitial changes occur in a late atrophic stage.

2 A chronic nephritis with atrophy, induration, and scarring. This condition originates in a specific arteritis and is not a primary disease of the kidney.

Dowling states that . . . "When one considers the march of events in syphilis it is not surprising that the response to the infection of a particular organ or tissue is generally more or less explosive in character in the early period, with a tendency to spontaneous resolution and, on the contrary, more sluggish in the late

Fig. 340—Retrograde pyelogram showing a markedly displaced left ureter which reaches almost to the right of the spine at the fourth lumbar vertebra. The whole system of calices is grossly elongated; some of the calices appear to be obliterated, others were dilated and deformed.
(Courtesy of Dr. Archibald W. Hunter and *Journal of Urology*, 1939, 42 1176.)

stages with little or no tendency to recovery without treatment. One would expect, therefore, an acute nephritis of comparatively short duration in the early stage, and in the later stages a subacute or chronic type which, untreated, would run a protracted course and this, as a general rule, is found to be the case."

During the general invasive period, for a few weeks following

the appearance of the chancre, the invaded tissues develop a high degree of sensitivity and resistance to an unaccustomed infection. The secondary manifestations representing "a culminating explosive reaction of the hypersensitive tissues" result in the destruction of great numbers of spirochaetes, with the apparent semblance of having overcome the disease. It is the opinion of many authorities that no further spirochaetal invasion occurs after the secondary stage has passed. It is also believed that tertiary manifestations result from renewed activity of the spirochaetes which survived the first general reaction against them. However, the reaction against these secondary invaders is of a different quality; the infection is destroyed much more slowly and with greater difficulty. The superficial skin manifestations are replaced by gummata which are highly resistant to treatment. In the kidney, there is less explosive response to relighting a dormant infection than would be seen in nephritic involvement occurring during the period of systemic invasion. The nephritis resulting becomes a chronic process, runs a protracted course that cannot be distinguished from other forms of chronic nephritis.

Symptoms and Diagnosis: The classical subjective symptoms of syphilitic nephritis are edema, massive albuminuria, and backache. There is little or no headache. The urine, besides the albumin, will contain an abundance of casts of several varieties. epithelial, granular, and waxy, and in addition, some fatty droplets. Two very significant factors are evident, the rapidity of onset just before or after the onset of the secondary eruptive manifestations and an early tendency to uremia. Of the greatest diagnostic importance is the fact that in spite of the massive edema and abundant casts, there is little impairment of renal permeability. The phenolsulfonphthalein test reveals almost normal percentages of elimination, even when the symptoms and condition of the urine suggest severe renal impairment.

Fournier states that there is another type of syphilitic nephritis that is unaccompanied by general symptoms and is of short duration, albuminuria being the only symptom. The condition lends itself readily to treatment, but there may be recurrences.

The symptoms of syphilitic nephritis, occurring in the tertiary period of the disease, are much less intense than those demonstrated in the acute stage. The symptoms may be so mild as to be completely overlooked and it may not be until an associated gummatous lesion is discovered that antisyphilitic treatment is instituted. The nephritis of this stage runs a chronic protracted course, showing no tendency to spontaneous resolution. There is little or no tendency to improve under the ordinary treatment of nephritis. Chronic syphilitic nephritis follows a benign course. In spite of the massive albuminuria, there is little vascular change. The blood pressure remains relatively normal.

The diagnosis is based upon the history, a positive Wassermann test, positive urinary findings, and occasionally, the therapeutic test. The history of a recent or present chancre is the important point in the history of acute syphilitic nephritis. In chronic nephritis, the history of a previous luetic infection is important. The urinary findings of massive albuminuria and abundant casts, yet normal phenolsulfonphthalein elimination are also important factors. Possibly the most striking feature of syphilitic nephritis is the prompt response to antisyphilitic treatment.

Cystoscopy: There is nothing pathognomonic in the appearance of the vesical mucosa in the presence of syphilitic nephritis. However, as a means of differential diagnosis and individual renal function tests, cystoscopy may be utilized to great advantage. Bilateral ureteral catheterization and collection of specimens for microscopic and individual renal function tests may be desirous.

Treatment: Once a positive diagnosis of syphilitic nephritis has been made, prompt antisyphilitic treatment should be instituted

immediately However, in the suspected cases, antisyphilitic treatment should be cautiously administered If good results follow such administration, treatment may be regularly instituted The response of syphilitic nephritis to antisyphilitic treatment is prompt The reverse is true in nephritis arising from any other cause

GONOCOCCAL PYELONEPHRITIS

Gonococcal pyelonephritis is a comparatively rare clinical entity Uhle, in 1931, found only 12 proven cases in a review of the literature He added one of his own Sisk and Wear, in 1936, added another, bringing the total to 14 cases proven by smear, culture, fermentation of dextrose, and serology May, in 1941, presented a case of gonococcal pyelonephritis complicating renal tuberculosis thereby bringing the total number of proven cases reported to 15

Undoubtedly, the scarcity of reports belies the frequency of occurrence of this condition as cystoscopy and ureteral catheterization are contraindicated in acute or subacute gonorrheal urethritis Gonococci prefer columnar epithelium for their growth and it is reasonable to assume that the type of epithelium lining the ureter and renal pelves is unfavorable for their growth The possibility of such a renal infection is suggested by a persistent pyuria Pus is observed in the second and third glasses of a three glass urine test and such pyuria is accompanied by tenderness at the costovertebral angle Although there is a possibility of an ascending gonococcal infection, it is believed that such infection is hematogenous in origin

Symptoms: The clinical picture of gonococcus infection of the kidney is in no way peculiar to any other infection of the kidney The infection ultimately results in an acute pyonephrosis

Diagnosis: The diagnosis is established by the history of a pre

vious gonorrheal urethritis and the finding of gram negative intracellular diplococcus in the ureteral urine. Strict and rigid technic must be maintained in ureteral catheterization to eliminate the possibility of carrying the infection from below upwards to the renal pelvis. The organism must be identified further by culture on selected media and proven by fermentation and serological tests. Differential identification must be made from the *micrococcus catarrhalis*, which appears similar to the gonococcus but which reacts differently on fermentation tests. The gonococcus ferments dextrose only.

Treatment: Eradication of the gonococcus infection of the lower urinary tract is the first requisite of treatment. Elimination of the infection of the kidney is best accomplished by ureteral catheterization and pelvic lavage. Nephrectomy is indicated if pyonephrosis is present. Such surgical procedure would be managed the same as a similar condition resulting from other causes.

ECHINOCOCCUS DISEASE OF THE KIDNEY

Echinococcus infection of the kidney is a condition of hydatid cyst formation caused by the dog tapeworm, *Taenia echinococcus*. The latter is a small tapeworm belonging to the Cestoidea (a class of worms parasitic in man and lower animals) of the phylum platyhelminthes (flat bodied worms, containing both sexual elements). The adult worm is approximately 2.5 to 5 mm. in length. It consists of a rounded head (scolex) surmounted by a crown of 30 to 50 hooklets and suckers for adhesion to the intestinal mucous membrane and a flat, elongated, ribbon-like body for three or four segments (proglottides) each of which, when mature, is bisexual.

The host of the *Taenia echinococcus* is the canine family; the dog, wolf, fox, or jackal. The eggs of the *Taenia echinococcus*, containing hooked embryos, are passed with the feces of these

animals The embryos develop in the digestive tract of an intermediate host a domestic animal (cattle sheep swine) or man When the embryos are carried into the alimentary canal of man or domestic animals their capsule is dissolved by the digestive juices and the hooked embryos are freed The freed embryos burrow through the wall of the intestine to enter the portal circulation Usually these embryos are carried to the liver which accounts for the high percentage of hydatid cyst formations in that organ However the embryos may be carried to the lungs or they may reach the main arterial circulation to produce hydatid cysts in any part of the body Invasion of the kidney is relatively rare

Deve estimates that in 75 per cent of instances the liver is the site of hydatid formation The incidence of involvement of the various organs is reportedly about the same wherever the disease is common liver 70 per cent lungs 10 per cent spleen 2 5 per cent kidney 2 per cent other abdominal organs 5 per cent muscle or cellular tissue 5 per cent brain 5 per cent and bone 1 per cent The disease is uncommon in America Only 31 cases have been reported in America although the disease is of common occurrence in Australia Argentina New Zealand Iceland Uruguay Northern Africa Southern Europe Syria has perhaps the largest incidence of occurrence as the disease is pandemic

Pathology The embryos form larval cysts (hydatid cysts) Once established the growth of these cysts is slow but continuous and relentless taking 10 to 15 years before symptoms are manifest In size renal hydatids vary greatly (Fig 311) They are usually spherical in shape affecting the lower pole of the kidney more frequently than the upper pole The cyst wall consists of two layers The outer wall or layer is white in color and presents a characteristic laminated structure The inner or germinal layer is granular It is in this material that fluid collects forming a cyst The fluid filling the cyst is colorless and sterile having a specific

gravity of 1.006 to 1.015. The fluid contains a toxic substance which may cause attacks of uticaria or produce toxic effects if the cysts are ruptured during removal. Buds arise along the germinal layer, some of which become hollowed, forming brood capsules in which little clusters of new scolices on stalks are produced. Some

Fig. 341—Primary echinococcus disease of the kidney. Gross specimen measures 17.8 by 11.5 by 7.5 cm. Sagittal section of kidney shows two main cysts, smaller one in lower pole, with their daughter cysts that completely occlude the renal pelvis but are contained within the renal capsule.
(Courtesy of Dr. William A Barrett and *Journal of Urology*, 1940, 44 389)

of the buds formed on the germinal layer develop into daughter cysts which become detached and float in the cavity of the mother cyst. New scolices may be produced within the daughter cysts (Fig. 342).

Livermore divides echinococcus disease of the kidney into two groups: (1) Open. Those that communicate with a calyx or the renal pelvis; (2) closed. Those that do not communicate with a

calyx or the renal pelvis. Craig and Brown believe that rupture into a calyx or pelvis is due to a secondary infection by pyogenic organisms. The growth of the cyst is eccentric, extending from the site where the embryo originally lodged in the cortex; the

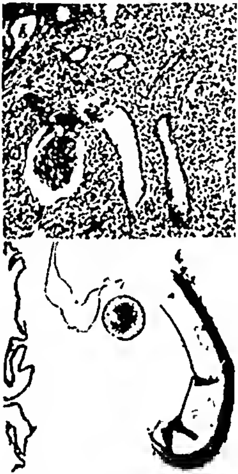


Fig 342—*Upper* Highpower photomicrograph through cyst wall and kidney tissue shows dense connective tissue with numerous lymphocytes, many eosinophiles and a few plasma cells. Renal tissue is heavily infiltrated by similar cells, there is an increase of connective tissue There are scattered foci in which leukocytes, chiefly eosinophilic, produce small abscesses.

Lower Sections through wall of cyst show a hemogenous chitinous material with a thin layer of cells on surface of this membrane In places those cells give rise to small bud like projections. No booklets are identified

(Courtesy of Dr William A Barrett and *Journal of Urology* 1940 44 389)

glomeruli and tubules gradually atrophy as the pressure is increased and the cysts enlarge.

Symptoms and Diagnosis: There are no pathognomonic symptoms. As the growth of a hydatid cyst is very slow, the cyst may reach considerable size before any symptoms are presented. The commonest complaints are pain in the renal area, frequency of urination, nocturia, and hematuria, none of which is pathognomonic. A carefully taken history, laying stress on the life habits and location of habitat, the close association with animals, pets or herds, may reveal important data.

Physical examination will usually reveal the presence of a tender mass in the renal region.

In the open variety, the finding of hooklets, scolices or laminated membrane in the urine is pathognomonic of hydatid cyst. However, some extrarenal focus may have ruptured into the urinary tract which would permit the finding of these typical structures. Confirmatory roentgenological findings of renal disease are imperative before surgical treatment of the kidney may be considered. X-ray examination usually shows an enlarged renal shadow. The pyelogram will show filling defects, distortion or even obliteration of the calices and possibly displacement of the pelvis. In the closed variety, pyelography will be of little diagnostic value.

Blood studies are important but are not totally accurate. Eosinophiles, although present in about 25 per cent of the cases, is not diagnostic of echinococcus disease. The Ghedini-Weinberg complement fixation test is of value but is not infallible. The test is positive in 84.3 per cent of cases according to Fairly. Casoni's intradermal injection of hydatid material is also of value. The test is positive in 92 per cent according to Dew, Kelloway and Williams. The greatest difficulty in this country in doing either the complement fixation test or the intradermal test is the lack of the

necessary antigens. Owing to the rarity of the disease, the antigens are not always available.

Cystoscopy: There is nothing characteristic or pathognomonic in the appearance of the bladder in echinococcus disease of the kidney. A small hydatid cyst may project from the ureteral orifice. If such a cyst is observed, a diagnosis may be made.

The finding of hooklets, scolices, or laminated membranes in the urine is the greatest aid in the diagnosis of hydatid disease of the kidney. The presence of these structures in the urine proves beyond question that hydatid disease of the urinary tract exists. It is then the problem to prove their source of origin.

Treatment: Treatment depends greatly upon the type of lesion present; that is, open or closed, and the extent and site of the pathological involvement. The concensus in this country is that nephrectomy is the best method of procedure, particularly in the closed type. Abroad, where the disease is more frequently observed, incision and drainage, or marsupialization of the kidney is preferred. The practice of aspirating the contents of the cysts through a large caliber needle or trocar is also widely advocated. Following aspiration, a similar quantity of ten per cent formalin solution is introduced into the cyst cavity.

ACTINOMYCOSIS OF THE KIDNEY

Actinomycosis of the kidney is rare. This fact is due to two reasons: (1) Actinomycosis as a disease is uncommon; (2) statistics have shown that the kidney is one of the rarest sites of the disease. Sanford and Voelker, in a survey of 670 cases of actinomycosis, found renal involvement in only one case. In the survey, they found only 45 cases of actinomycosis in children. Good studied 62 cases of actinomycosis of the abdomen and found secondary renal involvement in eight cases. The bladder was involved in three in-

stances. In one case, vesical fistula was found. Kretschmer and Hibbs, in a complete survey of the literature, found only three cases of renal involvement in children and added one of their own.

That the kidney is infrequently affected may be gained from an

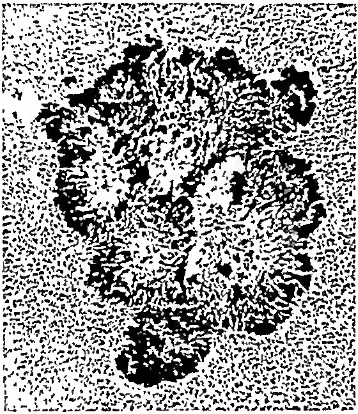


Fig. 343—Actinomycosis of the kidney. Microphotograph showing ray fungus in the tumor mass.
(Courtesy of Dr. Lloyd E. Kendall and *Urologic & Cutaneous Review*, 1934, 38:569.)

analysis of 628 cases of Ruhrah, who found that the head and neck were affected 357 times, or 55 per cent; the digestive tract, 132 times or 19 per cent; the lungs 92 times or 14 per cent; the skin, 16 times or 2 per cent; there were 33 doubtful cases, or 5 per cent (Fig. 343).

The causative organism is the *Actinomyces bovis.* This organ-

ism is a facultative anaerobe which is of a branching filamentous type. It is readily cultured and is easily killed by heat. Bollinger, 1877, described the condition as a distinct disease in cattle. Ponfick, 1879, demonstrated that the disease is identical in man and in cattle. Bostroem advocated a vegetable origin, but *Actinomyces bovis* has never been found in grains or grasses.

Actinomycosis is a chronic suppurative process characterized by sinus formation, the formation of granulation tissue and a brawny, leathery infiltration of the surrounding tissue. The pus contains the characteristic tiny yellow "sulfur granules." These granules, on microscopic examination, are seen to be a central mass of branching mycelia or filaments, with a definite radiation at the periphery and usually ending in well developed "clubs."

Edward concluded, after study of the possible sources of entrance into the human body, that *Actinomyces bovis* is a natural inhabitant of the mouth and gastrointestinal tract in man and animals. Garceau quotes Wright, who states that . . . "the *Actinomyces bovis* probably exists in the form of fragmental filaments growing in company with bacteria of the gastrointestinal tract." Israel also states that . . . "under certain conditions the malignant microörganisms may develop from the harmless leptothrix found in the mouth."

It may be impossible to demonstrate the site of entry of the actinomyces. It is doubtful if the kidney is the site of the primary infection. The kidney is usually considered the site of a secondary invasion, either as the result of a hematogenous metastatic infection, or the result of extension from a neighboring organ. The kidney shows multiple miliary globular areas scattered about in the kidney substance. These areas may be found singly or in groups. They are usually pale yellow but may be brown or green in color. As the colony grows, it excites an intense inflammation with round cell infiltration. Softening and abscess formation ulti-

mately result, the abscesses coalesce until an irregular abscess cavity is formed, in which the characteristic yellow granules are found. Surrounding the abscesses, a zone of granulation or connective tissue is formed. The tendency is not toward the formation of a large single abscess cavity, but rather the formation of many abscesses of small size. The abscesses are sharply demarcated from the surrounding parenchyma. Ultimately the process of abscess formation reaches the pelvis; evacuation occurs, following which the organism may be recovered from the urine. Usually the kidney is invaded by continuity from neighboring organs, most frequently the cecum. Adhesions are found between the kidney and the affected adjacent structures.

If dissolution of the tissues occur, an abscess cavity is formed and by sinus formation may extend to the external surface. Through the sinus may be discharged pus mixed with the characteristic yellow granules. The lesion affecting the kidney under such circumstances does not differ from the usual actinomycotic lesions occurring elsewhere in the body. There is thickening and erosion of the renal capsule. Following the erosion of the capsule, the parenchyma is invaded with the formation of characteristic suppurative lesions. Amyloid degeneration is the most frequent secondary degenerative change to which the kidney is subjected following such an infection.

Symptoms: There are no characteristic symptoms of renal actinomycosis. In those instances in which the kidney involvement is but a part of a generalized actinomycotic infection, the symptoms are those of a general sepsis. The symptoms referable to the kidney may not be of great intensity so that they may be completely overlooked.

In the primary form of kidney involvement, the symptomatology may be such as to be confusing with the similar symptoms found in pyelonephritis, pyonephrosis, tumor or tuberculosis.

The commonest symptoms are fever, loss of weight, and pain in the renal area. There is usually a leukocytosis. None of these symptoms is pathognomonic.

Renal actinomycosis rarely presents bladder symptoms. Good was able to find only three cases of vesical involvement in a series

Fig. 344—Retrograde pyelogram illustrating typical tumor deformity in right kidney. Note deformity in central part of right ureter.
(Courtesy of Dr. Lloyd F. Kendall and *Urologic & Cutaneous Review*, 1934, 38, 369.)

of 62 studied. These cases presented dysuria and frequency of urination.

Diagnosis. The diagnosis of renal actinomycosis is difficult. Such a diagnosis can only be established by the demonstration of the microorganisms in the urine or in the pus from a discharging sinus. Discovery of the fungus in the urine is conclusive evidence of the disease in some part of the urinary tract.

Cystoscopy is usually of little value. In rare instances, small ulcers may be found surrounding one of the ureteral orifices, but more frequently the picture of a generalized cystitis will be portrayed.

Pyelography has been of little value (FIG. 344). The picture presented has been confusing. In some instances, filling defects suggestive of renal tumor were observed; in other instances, renal tuberculosis was suggested. However, with the definite isolation of the microörganism and with abnormal findings at pyelography, the diagnosis is more than suggestive.

Treatment: Nephrectomy will usually effect a cure in cases of unilateral actinomycotic infection. If other organs are also involved, the chances of cure are very remote.

XXVI

RENAL CYSTS

Renal cysts are attached to but are not a part of the kidney parenchyma (PLATE LXXI). There are several types of renal cysts inherent in the kidney and they may be classified as follows:

(*a*) Retention cysts
(*b*) Solitary serous cysts
(*c*) Solitary hemorrhagic cysts
(*d*) Multilocular cysts
(*e*) Polycystic kidney

(*a*) RETENTION CYSTS

Small multiple retention cysts are of little clinical importance and are considered to be of acquired origin. The cysts are associated with the cicatricial changes of chronic nephritis. The cysts are usually very small, multiple, and located on the surface of the kidney.

(*b*) SOLITARY SEROUS CYSTS, (*c*) HEMORRHAGIC CYSTS, AND (*d*) MULTILOCULAR CYSTS

Solitary serous cysts of the kidney are usually large single cysts (FIG. 315). There is no structural difference between a solitary serous cyst and a solitary hemorrhagic cyst except the difference of

Examination

Tumor
Usually palpable in loin.

Urine
Usually negative.

Cystoscopic data
Bladder mucosa normal.

Meatoscopy
Orifice normal in appearance

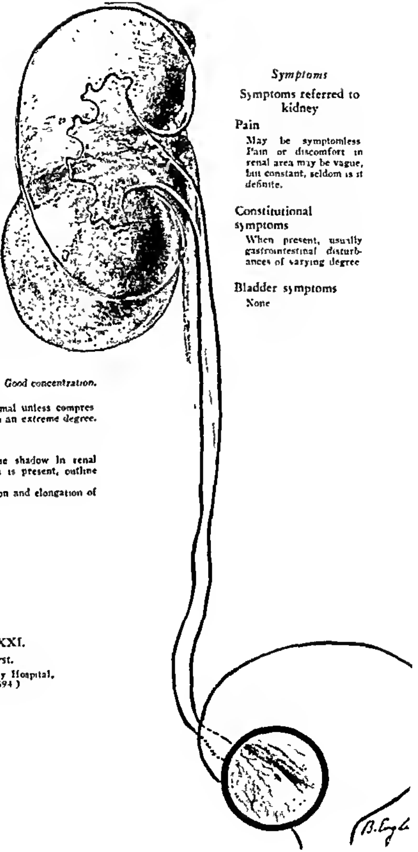
Function Tests

Phenolsulfonphthalein
Normal functional ability.

Indigo-carmine
Normal appearance time. Good concentration.

Functional tests are normal unless compression atrophy is present to an extreme degree.

X ray
Plain Large soft tissue shadow in renal area When calcification is present, outline of cyst readily seen.
Pyelography Compression and elongation of a calyx.



Symptoms

Symptoms referred to kidney

Pain
May be symptomless Pain or discomfort in renal area may be vague, but constant, seldom is it definite.

Constitutional symptoms
When present, usually gastrointestinal disturbances of varying degree

Bladder symptoms
None

PLATE LXXI.
Renal Cyst.
(Temple University Hospital, Acc. No. 2594)

the contents (Fig 346) The multilocular cysts are identical with solitary cysts except that the cysts are multiple They do not intercommunicate Solitary cysts are usually unilateral although there are instances reported where the condition was bilateral The size of the cyst may vary from one of small size to one containing several

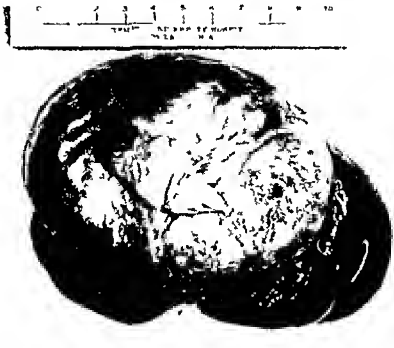


Fig 345—Solitary cyst of kidney The cyst with its calcified walls is considerably larger than the kidney

(L E McCrea American Journal of Surgery 193[illegible] 68 I R Temple University Hospital Acc No 39646)

liters of fluid (Fig 347) The condition is more frequently observed between the ages of 30 and 60 It occurs in both sexes but is observed more commonly in the female

The right kidney is more frequently involved than the left The lower pole is more commonly affected than the upper pole (Fig 348) The convex border of the anterior surface may also be the site of origin The serous cyst contains a clear, yellowish watery

fluid composed of albumin, chlorides, a few epithelial cells and leukocytes, and occasionally slight traces of urea. The interior of the cyst wall is lined by a layer of endothelium. The cyst usually arises from the cortex, but does not communicate with the renal pelvis. The wall is such an integral part of the parenchyma that

Fig. 346—Right kidney bisected longitudinally just after removal. From above downward is seen the normal upper pole and the thin-walled serous cyst and the thick-walled cyst filled with bloody fluid and fresh clots.
(Courtesy of Dr. J. Dellinger Barney and *Journal of Urology*, 1936, 36 602.)

enucleation is at times impossible (Fig. 349). Occasionally the walls of the cyst present calcareous deposits which form a thin, calcific shell (Fig. 350).

Solitary cysts are considered to be acquired in origin although many authorities consider the possibility of congenital origin.

Symptoms: A solitary cyst may not produce any symptom other than that of pressure on the adjacent organs. When present, the predominant symptoms are pain on the affected side, gastrointestinal disturbances of varying intensity. Hematuria, although uncommon, may be seen occasionally. In one case personally

observed, hematuria was the only symptom. Occurring spontaneously in a young man of 19 years, it was only after complete urologic study that a preoperative diagnosis of solitary serous cyst with calcification was made.

Fig. 347—Solitary cyst of the kidney. Intravenous urogram showing marked displacement of left kidney. At operation, the cyst was found to contain 1650 cc. of light colored fluid.
(Philadelphia General Hospital.)

Diagnosis: The diagnosis of solitary serous cyst of the kidney is seldom made preoperatively. Unless the cyst is large enough to produce a palpable mass, it may only be incidentally discovered during an exploratory operation. A plain roentgenogram may show the rounded outline of the cyst in approximation to the kid

ney shadow. Unless the cyst exerts considerable pressure on the pelvis of the kidney, the pyelographic appearance is entirely normal. It is usual that the pyelogram is normal when the cyst is attached to the upper or lower pole of the kidney, but if the cyst

Fig. 348—Solitary cyst of kidney. Retrograde pyelogram. The inferior calyx is elongated by pressure by the cyst, the walls of which show calcific deposits.
(L. E. McCrea, *American Journal of Surgery*, 1943, 60 328
Temple University Hospital, Acc No 39646.)

is attached to the middle of the kidney, it is probable that pyelography will show a filling defect of the pelvis of the kidney. Such a defect may be misleading and suggest the possibility of neoplasm or polycystic kidney.

Cystoscopy: There is nothing pathognomonic in the cystoscopic appearance of the bladder in solitary cyst of the kidney. Possibly

Fig. 349—Microphotograph showing the dense hyalinized fibrous cyst wall and the contiguous compressed and elongated kidney tubules.
(L. E. McCrea *American Journal of Surgery* 1943, 60 378
Temple University Hospital Acc No. 39646.)

the greatest value of cystoscopy is normal renal function as demonstrated by individual renal function tests and the presence of normal ureteral urine. Retrograde pyelography, done at the time of cystoscopy, is possibly the greatest diagnostic aid.

Treatment: Resection of the cyst together with a portion of the kidney is recommended whenever possible. Nephrectomy is to be avoided except in those instances of marked destruction of the kidney by the cyst or other associated diseases.

Fig. 350—Simple cyst with calcification. The kidney is bisected longitudinally. Note the normal upper pole and the thick, fibrous wall of the cyst. The cyst was filled with cellular debris impregnated with calcareous salts.
(Temple University Hospital, Acc No. 3767)

(*e*) POLYCYSTIC KIDNEY—CONGENITAL CYSTIC KIDNEY

Polycystic kidney disease is an extensive replacement of normal renal parenchyma by cystic formations (PLATE LXXII). The condition is congenitally bilateral but may progress more rapidly on one side than the other. The kidneys are usually larger than normal and are sometimes enormous. Instances of tremendous enlargement have been noted in the fetus at birth, as well as in later life. In instances of extreme enlargement in the fetus, the polycystic kidneys may be so enormous as to impede delivery. Usually,

the fetus dies *in utero* or very shortly after birth The appearance of a polycystic kidney is characteristic and cannot be mistaken for any other condition (FIG 351) The affected kidney may present a variety of colors gray in various tones, different shades of yellow,

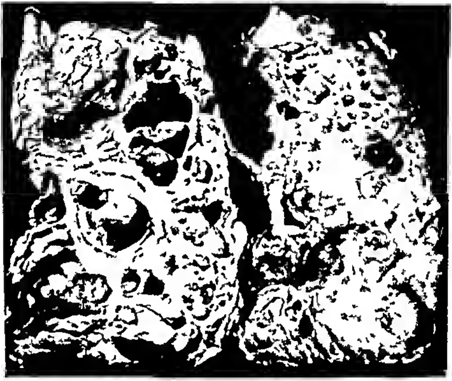


Fig. 351—Polycystic kidney The kidney is enlarged with loss of its characteristic shape *Multiple cysts of varying sizes replace the parenchyma.*
(Temple University Hospital Acc No 346)

red or blue The cysts vary in size from minute vesicles to cysts several centimeters in diameter The cysts may be so *closely* packed together that the kidney appears to be entirely composed of cysts of varying sizes On section the renal tissue may be seen to exist between the cysts The renal tissue usually shows progressive changes of atrophy from pressure and interstitial fibrosis

Polycystic disease of the kidney is uncommon Naumann in a

Examination

Tumor

Usually palpable in both loins.

Urine

Red blood cells—varying amounts. Usually low specific gravity

Cystoscopic data

Bladder mucosa normal.

Meatoscopy

Orifice normal in appearance. Urinary efflux may show blood.

Function tests

Phenolsulfonphthalein

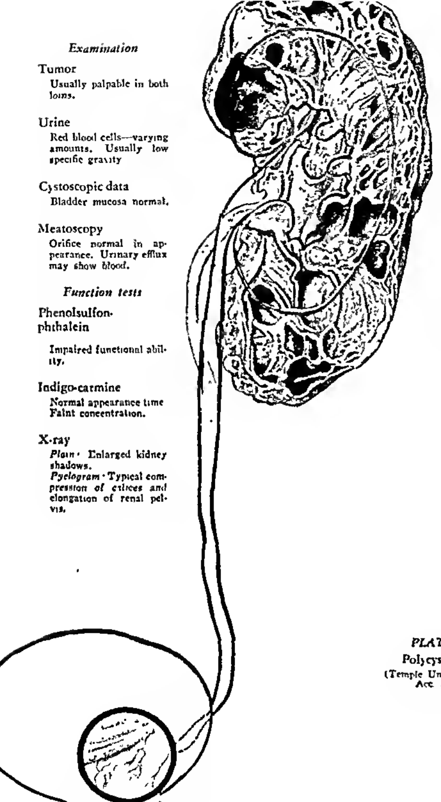
Impaired functional ability.

Indigo-carmine

Normal appearance time Faint concentration.

X-ray

Plain: Enlarged kidney shadows.
Pyelogram: Typical compression of calices and elongation of renal pelvis.



Symptoms

Symptoms referred to kidney

Pain

Usually not intense, but when present usually confined to lumbar region. Occasionally passage of blood clots produces typical colic

Hematuria, in varying degrees, very frequent

Constitutional symptoms

Usually those of renal insufficiency, such as nausea, vertigo, anorexia, headache, lassitude loss of weight, are common

Bladder symptoms

None

PLATE LXXII.

Polycystic Disease.

(Temple University Hospital, Acc. No. 4353.)

summary of 10 177 autopsies found such a condition 16 times 14 of these were bilateral The ages observed vary from the newly born infant to advanced age Sieber in an analysis of 211 reported cases found the age incidence to be as follows

8 weeks to 19 years	32 cases
20 years to 29 years	26 cases
30 years to 39 years	22 cases
40 years to 49 years	67 cases
50 years to 59 years	40 cases
60 years to 69 years	10 cases
70 years to 79 years	6 cases
80 years to 89 years	2 cases

In 12 029 autopsies at the Philadelphia General Hospital the author found the condition to exist in 16 proven instances From the years 1936 to 1941 inclusive, in 151 223 admissions a clinical diagnosis of polycystic kidney was made 11 times The ages observed varied from the newly born infant to advanced age

It can readily be seen that the predominant age of occurrence is in the young and between 40 and 60 years of age

There is a marked hereditary tendency A familial history is of considerable diagnostic value In a case observed and studied by the author the patient a young man of 28 years presented a history of albuminuria and hypertension which varied between 168/120 and 158/108 On physical examination both kidneys were palpable Urea nitrogen was 24 and 26 mg per 100 cc Specific gravity was fixed at 1 006 There were from 25 to 30 red blood cells per field Familial history revealed that his mother three sisters and two brothers suffered with kidney pathology Two sisters were known to have polycystic kidney and another had tuberculosis of the kidney

Etiology: The etiology of polycystic kidney disease is obscure. Its presence is considered to be of congenital origin due to occurrence of the condition in the newborn and the fact that it is bilateral. It is frequently associated with cystic changes in the liver and spleen. The hereditary tendency is recognized. Of all the

Fig. 352—Photomicrograph showing the histological appearance of polycystic kidney.
(Courtesy of Dr. Lawrence W. Smith and Dr. Edwin S. Gault.)

theories advanced for the occurrence of polycystic renal disease, none has been universally accepted as proving its pathogenicity.

Pathology: The appearance of a well advanced polycystic kidney is typically characteristic. Although retaining the shape of a normal kidney, a kidney so affected is enlarged or may be enormous. The condition is bilateral although one kidney may be of normal size when inspected at operation, but which will rapidly present a typical appearance if the opposite or definitely known

polycystic kidney is removed. The cysts vary in size from minute vesicles to cysts several centimeters in diameter. They are discrete and individual, do not communicate with each other or with the kidney pelvis. The cysts are distributed through the renal parenchyma (FIG. 352). The walls of the cysts are thin and occasionally rupture into the renal pelvis, which would account for the presence of hematuria. The cysts are usually clear or semitransparent and contain a clear yellowish fluid which varies considerably in its contents. The cysts have been reported to contain calcium oxalate, uric acid, cholesterol, leucin, cystin, fat, blood and pus, hippuric acid, and tyroleucin.

Symptoms: The infants so affected usually die within a short time. In the adult, the cases encountered may be divided into two groups:

Group I: Latent or symptomless group

The presence of polycystic kidney is accidentally discovered at operation for some other abdominal condition, or revealed at autopsy.

Group II. Active or symptom-producing group

These instances are manifested by a tumor formation and renal insufficiency. The picture generally presented is one of definite renal insufficiency, gradually increasing in intensity, with a palpable tumor in one or both loins.

Pain: Although apparent in many instances, pain is not a constant symptom. When pain occurs, it is of a dull, continuous character in the loin. Hematuria also occurs in relatively one third of the cases The hematuria does not differ from hematuria of any other cause. The clinical picture is one of chronic nephritis and renal insufficiency. Headache, nausea, vertigo, anorexia and loss

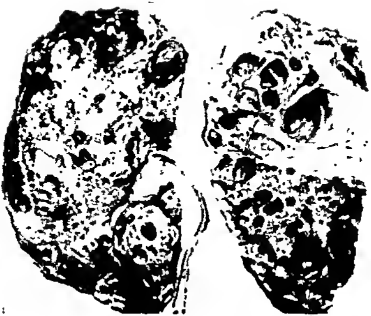


Fig. 353—Left, exterior of kidney; right, cut section, the white area at upper pole was tuberculous.
(Courtesy of Dr Charles F. Engels and *Journal of Urology*, 1940, 44 873.)

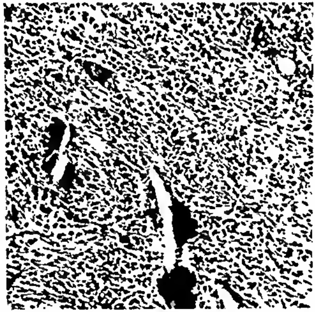


Fig. 354—Tuberculosis in polycystic kidney. Section from tuberculous area.
(Courtesy of Dr Charles F. Engels and *Journal of Urology*, 1940, 44 873.)

of weight are common. These symptoms are usually associated with an elevation of the blood pressure of moderate extent only. The eye grounds present the typical appearance commonly seen in chronic nephritis. As in chronic nephritis, there will be urea nitrogen retention and the dye elimination tests will usually be

Fig. 355—Hypernephroma in polycystic kidney. Low-power photomicrograph showing area of tumor within polycystic kidney.

(Courtesy of Dr. M. M. Melicow and Harold H. Gile and *Journal of Urology*, 1940, 43: 267.)

observed as lower than normal. Any other form of kidney disease may be present such as tuberculosis (Figs. 353, 354), tumor (Fig. 355) or pyelonephritis, in which event the symptomatology of the complication is usually the most pronounced. In other instances, there may be no other urological symptomatology than that presented by a chronically advanced glomerular nephritis. The

symptoms may be apparent for months or even years, with associated renal dysfunction, anuria, uremia and ultimate death.

Diagnosis: The diagnosis of polycystic kidney disease can only be made by the interpretation of the physical findings, the symptomatology, and by the pyelogram (Fig. 356). The symptomatology may be such that the condition may be considered an interstitial nephritis. Both kidneys are usually palpable; occasionally one kidney is palpable, while at other times, neither kidney is palpable.

Fig. 356—Polycystic kidney. Bilateral retrograde pyelogram revealing the abnormal position of the kidneys and the elongated "spider like" deformity of the calices.

(Philadelphia General Hospital.)

Cystoscopy: There are no pathognomonic features observable

a cystoscopic examination as would establish a diagnosis The mucosa of the bladder is usually normal in appearance Occasionally a moderate degree of congestion of the trigone may exist Differential phenolsulfonphthalein tests show delayed appearance on both sides and a diminution in the percentage output

The ultimate diagnosis of polycystic kidney rests mainly upon the roentgenological findings As a rule the pyelogram presents a typical appearance The narrowed pelvis and elongated calices extend over an area greater than that of the normal kidney There are many variations The pyelogram may even simulate renal neoplasm In the earlier stages the picture presented shows a crescentic distortion of the terminations of the minor calices There is elongation of the major calices with associated widening and cupping of the minor calices The picture presented by the pyelogram will often vary with the stage of the disease Due to the frequency with which this condition is noted on both sides a bilateral pyelogram is contraindicated due to the frequency with which the diseased kidney undergoes a transitory anuria following such a study It is deemed wise to assume the conservative attitude of doing one side permitting a period of several days to elapse before attempting pyelography of the opposite side A study of both sides should always be done if such renal pathology is considered

Prognosis The prognosis of polycystic kidney is poor although many patients may be carried along by proper diet and rest for a period of many years with impaired renal activity The kidneys so affected are more subject to infection with its associated symptomatology than the normal kidney

Treatment The treatment of congenital polycystic kidney is medical rather than surgical The patient should be maintained on an alcohol free diet low in proteins A high fluid intake should be maintained In the event of complicating renal pathology affecting one side surgical treatment should lean toward conserva

tive management rather than to the radical. Much attention should be paid to the excreting ability of the opposite kidney before surgical measures are instituted.

HYDROCELE RENIS

Hydrocele renis (perirenal hydronephrosis, renal hygroma, pseudohydronephrosis, hydroperinephrosis) is a rare condition. Kelley, in 1903, is credited with the first description of the condition. Renner, in 1923, applied the term "Hydrocele Renis."

The origin of the fluid is controversial, although there are three possible sources from which the fluid may be acquired; renal, hemovascular and lymphovascular. The fluid contained within

Fig. 357—Hydrocele renis. Gross specimen showing complete detachment of capsule from kidney surface which is grossly normal in appearance.
(Courtesy of Dr. Charles H. DeT Shivers and Dr. John H. Mathis and *Journal of Urology*, 1941, 46:1079.)

the fibrous sac may be urinous serous or serosanguinous The fluid surrounds the kidney partially or completely (Fig 357) The amount of fluid surrounding the kidney may vary There is no etiological factor of hydrocele renis but the condition may result

Fig 358—Hydrocele renis—Pyonephrosis Histologic section showing delicate layer of fibrin with enmeshed red cells covering denuded surface of kidney Note interstitial exudate between tubules within cortex

(Courtesy of Dr Charles H DeT Shivers and Dr John H Ma[illegible] and Journal of Urology 1941 46 10 9)

from one or more factors renal trauma perirenal inflammation lymphatic obstruction or urinary obstruction (Fig 358)

Symptoms The symptoms are usually not acute *The first symptom* is usually a sense of fullness in the loin An abdominal or loin mass or tumor may be palpable Constipation nausea and vomit

ing from intestinal obstruction, or symptoms of biliary colic due to pressure in the common duct may be present.

Diagnosis: The diagnosis of hydrocele renis may be difficult due to the vague nature of the symptoms and indefinite physical findings. Renal function may vary from normal to complete lack of function depending upon the involvement of the renal parenchyma and free drainage into the bladder. Urinary findings are never suggestive.

Cystoscopy: There is nothing pathognomonic in the cystoscopic findings.

Pyelograms vary according to the presence or absence of hydronephrosis, compression of the renal pelvis and obstruction to urinary flow into the bladder. A definite diagnostic finding would be the pyelographic demonstration of a connection between the renal pelvis and the surrounding sac of fluid. Such a finding is seldom demonstrated.

Treatment: Nephrectomy is the procedure of choice.

XXVII

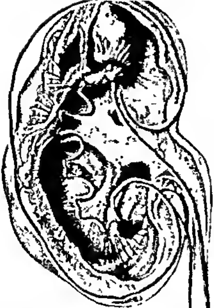
RENAL CALCULUS

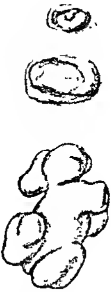
Etiology: The cause of renal calculus is not known (PLATE LXXIII) There are many theories regarding the etiology of renal calculus None of these theories, either alone or in combination, is sufficient in the explanation of calculous formation. Whatever the formation of calculi or stones in the urinary tract may be, their presence produces one of the major problems in urology. Although considerable recent research has been done and many suggestions have been offered for their dissolution within the renal pelvis, no known method or medication will satisfactorily dissolve them The calculus or calculi, having once formed, must either be passed or be surgically removed If calculi are permitted to remain within the kidney or renal pelvis, irreparable destruction of renal tissue may sooner or later occur (FIG 359).

The theories of calculous formation are:

Colloid crystalloid balance Considered to be highly significant in the formation of renal calculi The urine is regarded as a supersaturated solution, the colloid elements retain a large quantity of *crystalloids in solution* Disturbance of this balance permits crystal precipitation with beginning calculous formation.

Infection Infection undoubtedly plays an important role in certain instances of renal calculous formation Although calculi are known to be formed under aseptic conditions in certain instances it is usual that infection occurs following calculous formation Calculi have been produced experimentally by im-





Examination

Tumor

Usually not present Muscular spasm and tenderness at costovertebral angle

Urine

Microscopic blood usually found during and immediately following attack of renal colic. Usually moderate number of leukocytes In infected cases, pyuria.

Cystoscopic data

Bladder mucosa normal.

Meatoscopy

Varies with position of calculus If high up in the ureter the ureteral orifice is normal.

If just above the orifice, marked edema and hyperemia If a *small calculus has been* passed, considerable swelling and ecchymosis around the orifice will be present

Function tests

Phenolsulfonphthalein

Delayed, impaired function.

Indigo-carmine

Delayed appearance time. Faint concentration.

X-ray

Plain In 90 95 per cent of instances, calculi are radiopaque
Pyelography Shows calculus in relation to renal pelvis.

Symptoms

Symptoms referred to kidney

Pain

Recurrent attacks of renal colic. Sudden, lancinating pain in renal re*gion, radiating downward* along course of ureter to groin, bladder, genitalia

Constitutional *symptoms*

Nausea and vomiting on occasion Marked weakness and exhaustion following attacks.

Bladder symptoms

Frequency of urination very common At times, excessive

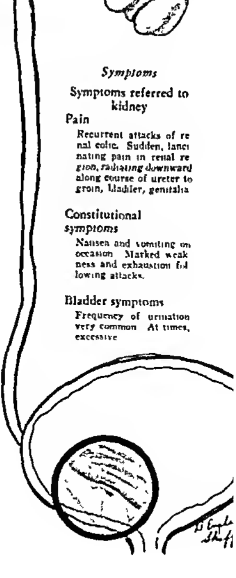


PLATE LXXIII.

Renal Calculus.

(Temple University Hospital, Acc. No. 3807)

Fig 359—Hydronephrosis secondary to multiple calculi.
(Temple University Hospital Acc No 3807)

plantation of streptococci removed from the infected teeth of patients suffering with recurrent lithiasis into the devitalized teeth of dogs It is apparent that infection plays some role in the formation of renal calculi Various strains of staphylococci, streptococci, *Bacillus coli* and the *Proteus ammoniae* are the bacteria most frequently encountered in the presence of calculi It is noteworthy to observe the frequent association of lithiasis in the presence of

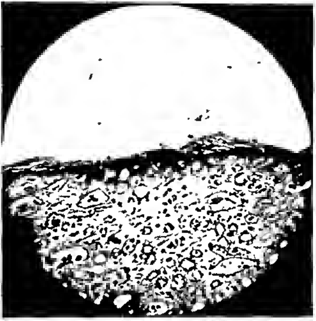


Fig. 360—Photomicrograph through a typical plaque of calcium showing distinctly under the normal covering epithelium. Special staining methods have proven this to be calcium. Note the absence of evidence of infection.

(Courtesy of Dr Alexander Randall and Dr Perry D Melvin and *Journal of Urology*, 1937, 37 737)

chronic suppurative wounds such as are seen in osteomyelitis. The mechanism or the role performed by infection in calculous production is not clear and is not understood. Randall believes that infection plays an important role by producing minor papillary lesions or injuries to the calyceal linings. These infection-induced injuries constitute focal points where crystallization of urinary salts may occur with the production of a primary renal calculus (FIGS. 360, 361, 362).

Stasis: Stasis of the urinary flow is not a cause of calculous formation. An important factor of calculous formation is incited by the stagnation of the urinary flow with an associated infection which invariably occurs. The frequency of occurrence of calculi in the presence of congenital renal anomalies is undoubtedly favored by

stasis of the urinary flow. Similarly, the acquired obstructive uropathy causing stasis can hardly be considered a direct contributory factor of calculous formation. Stasis of the urinary flow cannot be productive of renal calculi. Many other factors in association are necessary.

Metabolic disturbances: The role of calcium metabolism is of

Fig. 361—Photomicrograph through specimen. Here the epithelium covering the calcium plaque has gone and the plaque lies raw and bathed in calyceal urine. The wrinkled outer covering is of different character and coloring from the supporting plaque, and appears to be a distinct secondary deposit. Note the early calcium deposits under the epithelial cells lining the uriniferous tubules in the deeper portion of the tissue, and the absence of any evidence of infection. This is the earliest evidence of the beginning of a renal calculus yet observed.
(Courtesy of Dr. Alexander Randall and Dr. Perry D. Melvin and *Journal of Urology* 1937, 37 737.)

particular importance in the production of renal calculus. There are few calculi found in the urinary tract in which calcium is not found in one or more of its many salts. It has been shown that a diet deficient in vitamin A will produce calculi in rats. It has been suggested that calculous formation results from a secondary disturbance of calcium metabolism rather than the direct result of dietary deficiency. Faulty calcium metabolism is evidenced in hyperparathyroidism. In this condition there is an increase of the

calcium in the urine. It has been shown that abnormally increased concentration of the calcium salts in the urine predisposes to calculous formation (FIG. 363).

Characteristics of Renal Calculi: Renal calculi vary widely in size, shape, color, structure, and composition. Simple or homogeneous calculi are composed of a single chemical substance. Mixed or

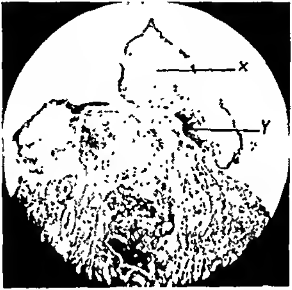


FIG. 362—Photomicrograph through the calculus. The calcium plaque is indicated by Y and the calculus by X. Note the loose attachment of the plaque. By differentiating stains the calculus has been proven to be calcium phosphate and the plaque a different salt of calcium.

(Courtesy of Dr. Alexander Randall and Dr. Perry D. Melvin and *Journal of Urology*, 1937, 37:737.)

heterogeneous calculi are composed of more than one substance. Calculi may be single or multiple in number. They may be unilateral or bilateral in position (FIG. 364). In size, renal calculi may vary from minute masses of agglutinated crystals, termed gravel, to large solitary solid stones, occupying practically the entire kidney and weighing many grams. The composition of renal calculi varies greatly. The usual compositions are:

Uric acid calculi are yellowish brown to reddish brown in color, are hard but fragile in consistency, round or ovoid in shape. The surface is smooth or finely granular. Uric acid stones are rarely pure in chemical composition but are usually admixed with urates or calcium oxalate.

Fig. 463—Parenchymal calculosis of kidneys. Autopsy specimen of both kidneys. Nephrostomy tube still *in situ*. Each stone has a brown-black nucleus.
(Courtesy of Dr. Maurice Muschat and Dr. L. Knolfe and *Journal of Urology*, 1939, 4, 793.)

Urate calculi are the most frequently observed stones in infancy and childhood. They are rarely seen in the pure form of ammonium urate but contain a mixture of phosphates or oxalates. These calculi are of a yellowish brown color and are relatively soft in consistency.

Oxalate calculi are usually composed of calcium or ammonium oxalate. They are very hard and are of dark brown color. These calculi may be exceedingly smooth or the surface may be covered with spines or spicula forming a "mulberry" calculus. Because of

their rough, irregular surface, they tend to be retained within the renal pelvis until they require surgical removal.

Phosphatic calculi are usually a mixture of calcium and ammonium or magnesium phosphate. These calculi occur either in an amorphous or in a crystalline state. They usually are multiple.

Fig. 364—Bilateral renal calculi. Roentgenogram—scout film with catheter *lying within the left ureter. Renal function poor.*
(Philadelphia General Hospital.)

They are usually of a grayish-white color, but may be yellow or brown. They present a rough, granular surface and are relatively soft and friable. The large coral or "staghorn" calculus, molded to the shape of the renal pelvis and calices, are generally of this chemical composition (Fig. 365).

Cystin calculi, in pure form, are smooth and polished but when mixed with phosphates they are rough and granular. Cystin calculi tend to assume the form of a calyx or pelvis and often form staghorn calculi (Fig 366). These calculi present a light greenish yellow color when freshly removed. The color darkens to

Fig 365—Large staghorn or dendritic calculus which occupied the entire renal pelvis causing total destruction of the kidney

a bluish green on exposure to light and air. These calculi are only slightly radiopaque and for this reason may be completely overlooked on roentgen examination.

Fibrin calculi are soft noncrystalloid concretions consisting of fibrin and calcium phosphates in alternate layers. They are usually small in size and multiple in number and may at times fill the renal pelvis (Fig 367). They are white, yellow or brownish in color and have a wax-like coating (Fig 368).

Rare stones: Carbonate calculi, occurring as calcium carbonate are occasionally seen. More rarely do they occur as magnesium carbonate. These calculi are chalky-white in appearance, crumble easily but cast a dense shadow to the roentgen ray.

Xanthin calculi present a yellowish-brown or grayish-green

Fig. 366—Cystin calculi. Roentgenogram showing multiple calculi. Patient, a boy of 12 years, excreted excessive amounts of cystin in the urine. (Temple University Hospital, Acc. No. 32528.)

color. These calculi are of a very hard, smooth consistency and rounded in shape. They cast a very poor shadow on the roentgenogram.

Indigo calculi are formed from indigo which may be derived from indican by oxidization. The calculi are of a bluish-black color. They are rare in occurrence.

Cholesterin calculi resemble cystin stones in appearance. These calculi are extremely rare in occurrence.

Urostealiths are related to fat and fatty acids (stearin and palmitin).

Bacterial calculi are round or oval, soft, elastic concretions, varying in size from 0.5 to 2 centimeters and are found in the renal

Fig 367—Soft calculi. Some were broken in handling with forceps (Courtesy of Dr Albert M Meals and *Journal of Urology* 1939, 42 1157)

pelvis These calculi are usually composed of *Bacillus coli*. Usually traces of urinary salts may be found in the concretions.

Symptoms: Renal calculi may exist for years without producing symptoms Symptomless calculi are not infrequently discovered during roentgenological examination for other conditions. It is the rule that in the majority of instances, symptoms are produced by calculi However, every symptom or group of symptoms re

sulting from calculi may be caused by other pathological renal lesions. In spite of the frequency with which renal calculi are seen, there is no single symptom or group of symptoms that may be considered pathognomonic.

The symptoms are most frequently pain, hematuria and pyuria. All these symptoms may be present or only one may be evident.

Fig. 368—Section of soft calculi showing laminations of amorphous debris, fibrin and crystals.
(Courtesy of Dr. Albert M. Meads and *Journal of Urology* 1939, 42:1157.)

Any combination of the symptoms may be complained of by the patient.

Pain: The pain caused by a renal calculus is usually a fairly constant, dull pain localized in the general region of the affected kidney (Fig. 369). The pain may be intermittent and may radiate. Radiation of the pain may be toward the genitalia, to the lower abdomen or toward the umbilicus.

Fixed renal pain may or may not be present. Such a pain is sel-

Fig. 369—Calculous pyonephrosis caused by a large impacted calculus at the ureteropelvic junction. Marked destruction of the kidney is evident. Pain was of an obscure nature localized in the renal area.
(Temple University Hospital, Acc. No. 3867.)

dom intense but is constant and annoying. Fixed renal pain is usually confined to the costovertebral area.

Renal colic. Renal colic is considered one of the most characteristic symptoms of kidney stone. The pain of renal colic is sometimes mild but it is usually severe. It is described as a stabbing, lancinating pain. The pain begins suddenly in the kidney region without premonitory symptoms and usually radiates to the groin, urethra or scrotum in the male, or the vulva in the female. The

radiating pain follows the downward course of the ureter to the bladder. Occasionally, retraction of the testicle occurs due to reflex spasm of the dartos or cremaster muscles. Immediately after the onset of the acute pain, the pulse will be accelerated, the skin cold and clammy. General abdominal rigidity is apparent during the attack of pain, relaxing completely as the pain subsides. Nausea and vomiting are common symptoms. An attack of pain may vary greatly in duration. An attack may be a brief spasm of a few seconds to a long-continued spasm lasting several hours or even days. If the pain is long continued, there are usually alternating periods of exacerbation of the pain and periods of comparative relief. Following an attack, there is usually constant muscle soreness in the renal area. Attacks of kidney colic vary greatly in frequency. The attacks may be a few days apart, or weeks, months or even years may intervene between attacks. The attack usually terminates as quickly as it begins.

Hematuria usually accompanies an attack of renal colic. Seldom is hematuria sufficient to produce clotting. The bleeding is usually only sufficient to present a "smoky" appearance of the urine. Massive hemorrhage is not indicative of stone but is more suggestive of neoplasm. The bleeding is usually microscopic in amounts. Hematuria may be continuous or may occur only after an attack of kidney colic, or it may appear spontaneously at intervals without pain or apparent cause.

Pyuria: The amount of pus in the urine may vary widely. Pus is absent in aseptic cases. Great quantities of pus in the urine are indicative of pyonephrosis. The amount of pus depends directly on the extent of infection. It may be absent if the ureter is totally occluded.

Diagnosis: A history of persistent pain in the loin or repeated attacks of colicky pain radiating along the course of the ureter

is suggestive of renal calculus. Hematuria or continued pyuria, or both in combination and the finding of crystals in the urine are also suggestive of renal calculus

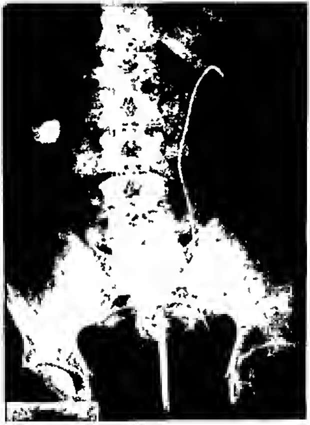


Fig. 370—Large solitary calculus lying in the renal pelvis of the right kidney (Philadelphia General Hospital)

Physical examination is usually of little aid in the diagnosis of renal calculus. All patients should be subjected to complete physical examination. An attempt should be made to discover any complicating condition that would preclude operation or vary any necessary operative procedure.

In making a diagnosis of renal calculus, the main reliance must

be placed upon roentgenographic demonstration (Fig. 370). It should be remembered that approximately five per cent of renal calculi are not demonstrable by the roentgen ray. The negative

Fig. 371—Calculous pyonephrosis. Intravenous urogram demonstrating a large calculus in the right kidney. Irregular collections of the contrast medium may also be observed.
(Philadelphia General Hospital.)

appearance of a plain x-ray film of the genitourinary tract does not preclude the possibility of calculous disease.

One of the most important details of study is intravenous urography. The roentgenograms should be exposed by stereoscopic procedure. Considerable informative data are to be gained by such study (Fig. 371).

1 The presence or absence of a calculus if opaque to the roentgen ray

2 The size shape and position of the calculus or calculi

3 The demonstration of partial occlusion of the ureter with subsequent distention of the renal pelvis Pelvic distention will be apparent even though the calculus is nonopaque

4 The position and excreting ability of both kidneys

5 The presence or absence of congenital anomalies of the upper urinary tract

6 The localization of renal and extrarenal shadows in exact relationship to the pelvis and calices of the kidney

All this information may usually be gained before continuing with the further studies of cystoscopy and retrograde pyelography and individual renal function tests

Cystoscopy Uncomplicated renal calculi present no evidence within the bladder that is suggestive of their presence The only observation that is suggestive of renal calculus is a blood efflux from the ureteral orifice on the affected side Such an observation is not characteristic of calculous disease (FIG 372) Such an observation may also be made in many other renal and ureteral lesions Occasionally a small calculus may be seen in the bladder a ureteral orifice showing evidence of recent trauma A small calculus may be seen projecting from the orifice of the ureter and is proof conclusive of the cause of the attack of pain In other instances edema surrounding the ureteral orifice is suggestive of a stone lodged in the intramural portion of the ureter It may be impossible or undesirable to catheterize the ureter *The ejection of* indigo-carmine from the ureteral orifice following intravenous injection will permit a rough estimate of the excreting ability of both kidneys and the patulousness of both ureters

By ureteral catheterization, the patency or position of obstruction within the ureter may be ascertained It is also possible to

ascertain the individual functioning ability of each kidney, as well as to collect specimens of pelvic urine for culture. It is also possible, by means of a wax-tipped catheter, to demonstrate the presence of a calculus which is nonopaque to the roentgen ray.

Fig. 372—Renal calculus. Roentgenogram showing a large dendritic calculus occupying the renal pelvis and calices of the right kidney.
(Philadelphia General Hospital, Acc. No 49650.)

Pyelography: Retrograde pyelography is the most important diagnostic procedure in the study of renal calculous disease. Such a study should include ureterograms as well as a pyelogram (Fig. 373). These procedures are the most valuable aids in the diagnosis of renal calculus available at the present time. Although the pyelogram is invaluable in differentiating between intra- and

extrarenal shadows, its great importance lies in the fact that pathological changes of the kidney may be readily visualized (Figs 374, 375) A kidney may not be visualized by intravenous urography because of some underlying pathological change preventing

Fig. 373—Calculous pyonephrosis. Retrograde pyelogram showing multiple calculi as well as marked destruction of the kidney
(Temple University Hospital Acc No. 40797)

normal excretory function such as hydronephrosis, pyonephrosis, tumors, tuberculosis, or anomalies

Investigation of the opposite kidney: Investigation of the opposite kidney should always be made before any estimation of treatment is instituted The estimation of the proper surgical

procedure will be greatly enhanced by a full knowledge of the functional ability of the supposedly normal kidney (FIG. 376). It is a recognized fact that the supposedly normal kidney is frequently the site of pathological lesions which may cause anuria

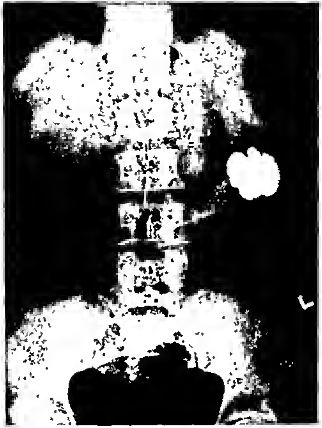


Fig. 374—Renal calculus. Roentgenogram showing large solid-appearing calculus.
(Temple University Hospital, Acc No. 49294)

to occur following renal operations. It has been repeatedly demonstrated that a calculus, with its typical symptoms, may be present on one side. On the other side, the kidney may be functionless as the result of other pathological changes such as tuberculosis, neoplasm or even calculous disease with massive

extrarenal shadows, its great importance lies in the fact that pathological changes of the kidney may be readily visualized (FIGS. 374, 375). A kidney may not be visualized by intravenous urography because of some underlying pathological change preventing

Fig 373—Calculous pyonephrosis. Retrograde pyelogram showing multiple calculi as well as marked destruction of the kidney.
(Temple University Hospital, Acc No 40297)

normal excretory function such as hydronephrosis, pyonephrosis, tumors, tuberculosis, or anomalies.

Investigation of the opposite kidney: Investigation of the opposite kidney should always be made before any estimation of treatment is instituted. The estimation of the proper surgical

procedure will be greatly enhanced by a full knowledge of the functional ability of the supposedly normal kidney (Fig. 376). It is a recognized fact that the supposedly normal kidney is frequently the site of pathological lesions which may cause anuria

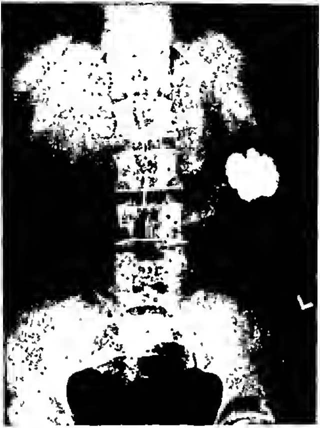


Fig. 374—Renal calculus. Roentgenogram showing large solid-appearing calculus.
(Temple University Hospital, Acc. No. 49294.)

to occur following renal operations. It has been repeatedly demonstrated that a calculus, with its typical symptoms, may be present on one side. On the other side, the kidney may be functionless as the result of other pathological changes such as tuberculosis, neoplasm or even calculous disease with massive

destruction of renal tissue The accepted practice in bilateral calculous disease is to institute surgical procedures primarily on the side functioning the most normally (FIG 377) All these highly significant and important data may be gained by the use of

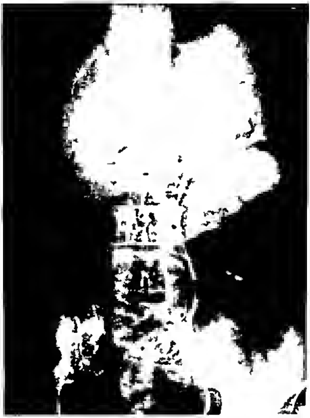


Fig 375—Retrograde pyelogram showing position of calculus in relation to renal pelvis At operation what appeared to be a solitary calculus was found to be a large collection of uniform calculi of small size
(Temple University Hospital Acc No 49794)

intravenous urography, cystoscopy with individual function tests, phenolsulfonphthalein or indigo-carmine, intravenously administered and retrograde pyelography

Anuria does occur following sudden, unilateral ureteral impaction with reflex suppression of the opposite and supposedly normal

kidney. The question is frequently raised whether or not a normal kidney can fail to function. It is generally conceded that suppression of urine occurs only in diseased kidneys, but that those lesions present within a supposedly normal kidney were never of such im-

Fig. 376—Renal calculus. Intravenous urogram showing a normally functioning left kidney. There is no apparent function of the right kidney. Multiple calculi of right kidney readily observed.
(Temple University Hospital, Acc. No 22469.)

portance as to produce symptoms. This fact is substantiated by a patient recently observed by the author. A man of 40 years presented himself with marked painless hematuria. Examination revealed the presence of a rapidly increasing tumor of the right loin. Within 24 hours, the tumor displaced the intraabdominal

organs toward the left and marked progressive anemia was evident No urine was passed in 24 hours Blood urea nitrogen was 145 mg, creatinine, 18 5 mg per 100 cc of blood An emergency nephrostomy was done, evacuating 2000 cc of bloody fluid from a

Fig 377—Bilateral renal calculi Roentgenogram showing a large solitary calculus in left kidney and multiple calculi in right kidney
(Philadelphia General Hospital)

hydronephrotic sac The output of the opposite kidney for the next 24 hour period was 3450 cc of urine of very low specific gravity A nephrectomy was later performed with an uneventful convalescence Subsequent study revealed the presence of a terminal phase of chronic glomerulonephritis that had never caused symptoms and was never known to exist The patient returned to

work but failed physically, dying of uremia in six months. Necropsy revealed a typical kidney of chronic glomerulonephritis.

Treatment: The treatment of renal calculus may be divided into two types: (1) Nonoperative; (2) operative. It is to the maintained end result that both methods lend themselves—a normally functioning kidney. It is common that when a patient is first observed it is for the relief of renal colic. The pain usually may be controlled by the hypodermic injection of morphine, 10 to 16 mg. ($\frac{1}{6}$ to $\frac{1}{4}$ grain) and atropin, 0.45 mg. ($\frac{1}{150}$ grain); or dilaudide, 2 to 2.5 mg. ($\frac{1}{32}$ to $\frac{1}{24}$ grain). The latter is preferred because of its greater ability in relaxing the spasm of unstriped muscle fibers. The undesired effects of atropin are avoided. Hot baths or a hot water bottle applied to the loin may cause muscular relaxation and relief of pain. If the pain continues for several days or recurs repeatedly after temporary relief, complete obstruction of the ureter may be present. It is possible, in some instances, to dislodge an occluding calculus by ureteral catheter. Occasionally, it is possible to introduce a ureteral catheter beyond a small calculus into the kidney pelvis and temporarily establish drainage as well as to dilate the ureter.

The specific treatment of the calculus or calculi depends on several factors: Size, position, whether calculi are unilateral or bilateral, age of patient, general physical condition, presence or absence of destructive lesions of one or both kidneys.

The size of the calculus and its relative position are very important factors. A calculus that is small enough to pass through the ureter into the bladder may be satisfactorily managed by ureteral dilatation. Discovery of a very small calculus lying in the calyx should not cause the patient to be subjected to immediate surgery. A small stone lodged at the ureteropelvic junction may pass following ureteral catheterization and dilatation. Cystoscopy should always be done to ascertain whether or not obstruction is complete

and whether or not infection is present following localization of a renal calculus by x-rays. If a very small calculus is found lying in a calyx and the renal pelvis is uninfected, the calculus may be permitted to remain without endangering renal tissue. If the pelvis

Fig 378—Renal calculus. Roentgenogram showing a calculus in the left kidney. The calculus was small but caused severe pain.

is infected and obstruction is evident, the calculus should be surgically removed. A small, silent calculus should be removed if at any time it begins to produce symptoms, or if it shows a tendency to enlarge, or if infection supervenes (FIG. 378). A calculus impacted at the ureteropelvic junction causing either partial or complete obstruction should be removed whether or not infection is present.

A great percentage of renal calculi are too large to pass through the ureter spontaneously (FIG. 379). Such calculi vary from a single pelvic stone approximately one centimeter in diameter, to the large, branched calculi completely filling the renal pelves and

Fig. 379—Renal calculus. Roentgenogram showing a large, solitary calculus in the left kidney.
(Temple University Hospital, Acc. No. 24068.)

calices. The calculi may be multiple, of the same relative size, or may vary in size. Calculi of such size, shape and contour should be removed to prevent kidney damage or to prevent further kidney destruction. Much depends on the general picture presented; the age, whether the calculi are unilateral or bilateral, and the relative

function of both kidneys. Huge bilateral dendritic calculi may exist for years with a low kidney function, yet these patients may continue indefinitely in a more or less normal way without operation. To subject these patients to operation would be unjustified in the presence of a high urea nitrogen, low kidney function and the relative age of the individual.

The one big problem of treatment of renal calculi is the maintenance of normal functional ability. Normal renal function cannot occur in the presence of obstruction. This statement is true whether calculi are unilateral or bilateral In the operating room more than anywhere else, conservative measures should be instituted in an attempt to conserve functioning renal tissue.

CALCULOUS ANURIA

Calculous anuria is the suppression of urine resulting from upper urinary tract lithiasis It is a condition that must be differentiated from the anuria produced by other causes. It must be further differentiated from the marked oliguria occurring in certain conditions of the kidney following ingestion of metallic poisons and in some phases of acute nephritis

Calculous anuria occurs because of:

(a) A single kidney (congenital or acquired) is obstructed.

(b) Both kidneys are obstructed.

(c) An acute obstruction is present on one side and a reflex suppression on the other side in an individual who possesses two supposedly normally functioning kidneys.

Cystoscopic study in nephritis or nephrosis would show an entire absence of obstruction or abnormality. In those instances of anuria due to calculus, definite obstruction should be demonstrated on one or both sides.

Symptoms: Calculous anuria may be asymptomatic but usually follows an acute attack of renal colic. Following the onset of anuria, the blood urea nitrogen and creatinine mount rapidly. The signs and symptoms of toxemia develop, which are proportionate to the increasing blood urea nitrogen and creatinine levels. Symptoms of uremia usually occur gradually, but steadily increase in intensity. Unless the obstruction to the urinary flow is eliminated, uremia increases in intensity and death ensues.

Diagnosis: The diagnosis of calculous anuria is made by bilateral ureteral catheterization and the roentgen ray. The roentgenogram would reveal a calculus along the line of the ureter on one or both sides. These findings, together with the failure to obtain urine from the bladder, establish the diagnosis.

Treatment: Approximately 30 per cent of all instances of calculous anuria recover spontaneously as the result of relief of the obstruction, with or without the passage of the calculus or calculi. The remaining 70 per cent die as the result of uremia after varying periods of time, unless drainage is instituted by ureteral catheter or following the surgical removal of the offending calculus or calculi. Ureteral catheterization should be done in an attempt to relieve the obstruction to the kidney and to establish drainage. Failure to catheterize successfully the ureter is a definite indication for surgical intervention.

CYSTINURIA

Cystinuria is a condition exhibiting abnormal amounts of cystin in the urine. Excessive cystin is possibly due to a congenital fault in the metabolism of sulfur containing amino-acids. According to Medes, cystin is excreted in small amounts by normal individuals. It was found, in a study of 50 normal subjects, that the variation in the daily output of cystin was from 0.8 to 81

mg In cystinuria, elimination of cystin in amounts far in excess of the normal are found in the urine Three or four times the maximum normal elimination may be found

Bodansky believes that a large proportion of cystin is derived from food and is required for the synthesis of hair and epidermal structures Human hair was found to contain 15 6 to 21 2 per cent of cystin He also states that cystinurics continue to excrete cystin even on a protein free diet and during starvation, which would indicate that cystin is at least partly endogenous in origin Others believe the amount of cystin present in the urine varies with the amount of protein ingested

Cystin in the urine, either in solution or in crystals, does not present any clinical symptoms The clinical picture presented is one of urinary lithiasis in those instances where the process has continued from crystallization to calculous formation Calculous formation is relatively rare Randall considers that 2 7, Hinman, 3 to 5 per cent of all cystinurics form calculi These calculi are not radiopaque unless admixed with calcium salts (FIG 380) For that reason, demonstration of cystin calculi may be difficult, if not impossible

Cystinuria is a condition of strong familial tendency It is a hereditary disease and is known to be transmitted by either parent to children of either sex A boy of 12 years observed by the author presented marked pyuria He was found to excrete 192 mg of cystin in a 24 hour urine specimen of 1325 cc X rays revealed the presence of multiple calculi in the renal pelvis Analysis of the *calculi, after removal revealed that they were composed of cystin* with a thin coating of calcium oxalate and phosphate Following operation, two cystin determinations were made Both determinations revealed a high percentage of cystin excretion, 183 2 and 296 mg respectively The family history was interesting since it demonstrated the familial tendency of cystinuria The father was

45 years of age and in apparently good health. The mother, 33 years of age, was in good health. The patient had three sisters; fourteen, nine and six years of age respectively. The father and eldest sister were found to be cystinurics but were without symp-

Fig. 380—Cystin calculi. Intravenous urogram showing the presence of calculi as well as destruction of the kidney. Cystin calculi proved by chemical analysis following removal.
(Temple University Hospital, Acc. No. 32528)

toms. The other members of the family were negative to all tests.

Diagnosis: The finding of both cystinuria and urinary calculi in the same individual suggests that the calculi are composed of cystin. The same principles of diagnostic procedure should be applied as in any other calculous formation. The visualization of the calculi may be somewhat difficult on occasion. Pure cystin

calculi are relatively invisible or cast a faintly visible shadow on the roentgenogram unless they are large or contain inorganic salts

Treatment: The treatment of cystin calculi is surgical. Cystinuria cannot be cured, but in the majority of instances, it is controllable. The control of cystinuria is best accomplished by limiting the protein intake, forcing fluids and the oral administration of alkalies.

XXVIII

TUMORS OF THE KIDNEY

I

EPITHELIAL TYPE

Benign

1. Adenoma
   (a) Papillary
   (b) Tubular
   (c) Alveolar

2. Cystadenoma

Malignant

1. *Papillary carcinoma (of pelvis)*
   Grade I
   Grade II
   Grade III
   Grade IV
2. Squamous cell (of pelvis)
3. Hypernephroma (adrenal origin)
4. Adenocarcinoma (malignant nephroma)
   (a) Papillary type
      1. Clear cell
      2. Granular cell
   (b) Alveolar

II

MESOTHELIAL TYPE

Benign

1. *Connective tissue origin*
   (a) Fibroma
   (b) Lipoma
   (c) Chondroma
   (d) Osteoma
2. Myoma
   (a) Leiomyoma
   (b) Rhabdomyoma
3. Angioma

Malignant

1. *Sarcoma*
   Fibrosarcoma
2. Myosarcoma
   (a) Leiomyosarcoma
   (b) Rhabdomyosarcoma

III

MIXED OR TERATOID TUMORS

No Benign	*Malignant*
	Wilms tumor (Embryoma or Adenomyosarcoma)

The large majority of all new growths of the kidney are malignant If untreated these tumors are more or less rapidly fatal It is therefore imperative to make an early diagnosis and to institute treatment before metastasis occurs (PLATE LXXII) In the majority of instances metastasis is relatively late but when it occurs it is usually widespread Even with our modern diagnostic methods an early diagnosis is the exception as the tumor growth is usually well advanced before symptoms are sufficient to attract attention The clinical manifestations of renal tumor may vary greatly Symptoms may be present or absent The symptoms may be vague and indefinite or they may be definite and positive There are no precancerous or characteristic early symptoms of renal neoplasm known to exist Death has been known to occur without clinical evidence of renal tumor even though widespread metastasis was present Permanent cure of renal neoplasm rests entirely with making an early diagnosis before metastasis occurs and the immediate institution of radical treatment

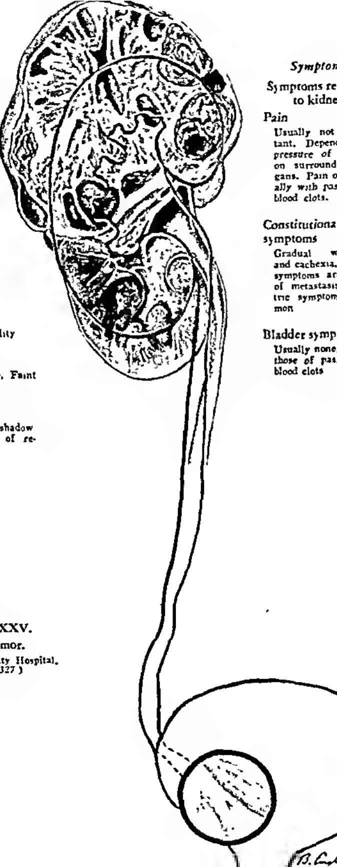
Few surgical conditions have been subjected to more controversy and misinterpretation than the pathological classification of renal tumors Confusion in the classification of these tumors has existed for years yet there is no comprehensive uniform view of the subject Much of this confusion of terminology of renal tumors may be attributed to the embryological development of the kidney which undoubtedly contributes greatly to the nature of many renal tumors

PLATE LXXIV—*Renal hematuria. Bloody urine may be seen to be ejected from a normal appearing ureteral orifice. A classical finding in renal tumors.*

Symptoms There is no single symptom or group of symptoms that is characteristic of renal tumor This is evident whether the tumor arises from the parenchyma or the mucosa of the renal pelvis The classical triad of symptoms usually observed hematuria tumor and pain *occurring singly or in combination* are the cardinal symptoms of renal tumor

Hematuria is the most frequent initial symptom of renal tumor Hematuria occurs in over 60 per cent as a first symptom in those persons so affected Hematuria is possibly the most constant and outstanding symptom of renal tumor In adults hematuria is usually the initial symptom and appears at such a time that the tumor is not yet palpable In children suffering with Wilms tumor a tumor is usually palpable before hematuria occurs Usually the bleeding of renal tumor is symptomless that is it occurs without pain or without apparent cause The bleeding in most cases is usually continuous for a period of a few hours or days and usually disappears as spontaneously as it occurred This is unfortunate for the patient from the standpoint both of early recognition and of treatment All too frequently is such a symptom disregarded by the patient who postpones examination or consultation until a later time The time selected by the patient for examination is usually too late for curative treatment

There is a great variation in the severity and duration of the hematuria The bleeding is always intermittent and seldom is it so profuse as to be dangerous It is usually considered that the amount of blood in the urine is greater in the presence of renal tumor than in any other form of renal pathology capable of causing hematuria Intense fatal hemorrhage is of very infrequent occurrence A single hemorrhage may be followed by weeks months or even years before recurrence of bleeding occurs Hematuria may be so slight that the urine is smoky in appearance or



Examination

Tumor
Usually present.

Urine
Blood in varying amounts.

Cystoscopic data
Bladder mucosa normal.

Meatoscopy
Orifice normal in appearance Urinary efflux may show blood

Function tests

Phenolsulfonphthalein
Impaired functional ability

Indigo-carmine
Delayed appearance time. Faint concentration.

X-ray
Plain Enlarged kidney shadow
Pyelogram Deformity of renal pelvis

Symptoms

Symptoms referred to kidney

Pain
Usually not important. Dependent on pressure of growth on surrounding organs. Pain occasionally with passage of blood clots.

Constitutional symptoms
Gradual weakness and cachexia. Often symptoms are those of metastasis. Gastric symptoms common

Bladder symptoms
Usually none, except those of passage of blood clots

PLATE LXXV.
Renal Tumor.
(Temple University Hospital. Acc. No. 4327)

the blood may be of microscopic amounts only. Usually the urine is dark red in color. Occasionally, clots of varying sizes may be passed through the urethra. The clots may be casts of the ureter and may cause pain from back pressure, or may produce colicky pain in passing down the ureter into the bladder.

Tumor. The presence of a tumor is usually a late manifestation of renal neoplasm in adults (PLATE LXXV). It is seldom that a tumor will be evident on inspection alone, but usually palpation is necessary to elicit its presence. In children, the presence of a tumor is the commonest initial symptom. Pressure exerted by a large tumor may be directed upward, causing respiratory embarrassment, downward, causing symptoms referable to the pelvic organs, or anteriorly, causing displacement of the spleen or colon. By palpation, it is possible to determine the consistency, the character of the surface, the mobility of the enlargement, together with the size and area occupied by the tumor.

Pain. Pain may be the most important symptom. It is considered an initial symptom in about 20 per cent of all cases of renal tumor. Usually, pain occurs late in the disease. However, pain is more likely to be due to metastasis than to the primary neoplasm. Pain in the renal area may be caused by the stretching of the renal capsule as the result of the enlarging tumor, or from hemorrhage within the renal capsule, or may result from pressure by the tumor mass on nerves or neighboring viscera. Colicky pain may occur during the passage of blood clots or obstruction of the ureter by blood clots.

Diagnosis. The early diagnosis of renal neoplasm is difficult, first, because of the insidious nature of the disease, second, the general laxity of both the profession and laity in their utter disregard of the importance of symptomless hematuria. The tendency to minimize its occurrence, coupled with the fallacy that hematuria,

having occurred only once, may never occur again, induces a false sense of security.

The diagnosis of renal tumor is based on:

1. Clinical history
2. Physical examination
3. Examination of the urine
4. Complete urological study
    (a) Cystoscopy
    (b) Renal function tests
    (c) Roentgenographic study
        1. Intravenous
        2. Retrograde

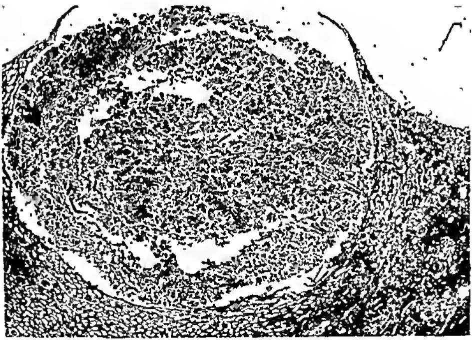


Fig. 381—Kidney—Adenoma. Low power microtessar photograph of an isolated *adenomatous encapsulated nodule.*

(Courtesy of Dr. Lawrence W. Smith and Dr. Edwin S. Gault.)

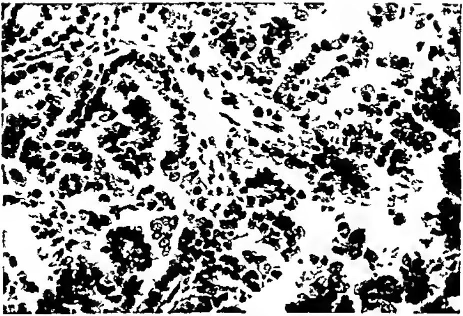


Fig 382—Kidney—Adenoma Highpower photomicrograph showing papillary arrangement of the rather characteristic small cuboidal cells which make up the majority of these lesions
(Courtesy of Dr Lawrence W Smith and Dr Edwin S Gault)

I

BENIGN EPITHELIAL TUMOR

Adenoma

Small renal adenomata are of little clinical importance They may grow to large size and may be productive of pain and hematuria When these tumors are large they are considered to be potentially malignant The small adenomata are discrete encapsulated sharply demarcated round grayish nodules situated in the cortex immediately beneath the capsule of the kidney (Figs 381 382) Adenomata may also occur in the medulla They may occur at any age and are found equally in either sex

Adenomata are essentially benign and are considered to be

derived from tubular epithelium or embryonal inclusions. Three types of adenomata are recognized according to the cellular arrangement (FIGS. 383, 384): (1) A papillary type; (2) a tubular type; (3) an alveolar type. Renal adenomata assume clinical importance only when they have grown so large as to produce pressure, or when they undergo malignant change (FIG. 385). Preoperatively, a differential diagnosis between a large, renal adenoma and a hypernephroma or other malignant tumor, is impossible.

MALIGNANT EPITHELIAL TUMORS

1, 2, *Renal Pelvic Tumors*

Tumors arising in the renal pelvis and calices are of an entirely different type or group than those tumors arising in the parenchyma. Tumors of the renal pelvis are observed less frequently,

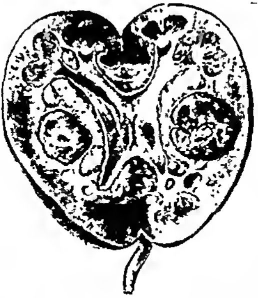


Fig. 383—Papillary cystadenoma of the kidney showing location of neoplasm.
(Courtesy of Dr. Norris J. Heckel and Dr. H. V. Gould and *Journal of Urology*, 1940, 44:200.)

Fig. 384—*Upper* Lowpower magnification showing marked variation of alveolar spaces which are lined by single layer of flattened cuboidal epithelium

Lower Highpower magnification showing presence of varying sized papillary protrusions into lumen.

(Courtesy of Dr Norris J Heckel and Dr H V Gould and *Journal of Urology* 1940 44 00)

Fig. 385—Malignant papillary cystadenoma of kidney. Appearance of kidney and tumor with right kidney beside it for comparison.
(Courtesy of Dr. Walter Gifford Hayward and *Journal of Urology*, 1940, 44 259.)

but the destruction of the kidney and the metastasis may be just as great as observed in parenchymal neoplasms Being epithelial in origin, these tumors present a similar pathological picture of the more frequently occurring vesical neoplasms (FIG 386) Tumors of the renal pelvis may be of papillary or of squamous cell variety It is considered that all such tumors are malignant or potentially malignant The papillary variety is observed more

Fig 386—Lowpower photomicrograph showing transitional cell papillary epithelial masses comparable to those seen in vesical tumors
(Courtesy of Dr Lawrence W Smith and Dr Edwin S Gault)

frequently than the squamous cell variety and occurs in relatively 75 per cent of instances of all pelvic tumors These tumors vary in differing degrees of malignancy, as do the papillary tumors of the bladder They may occur at any age, but are most frequently seen in men between 40 and 60 years of age Seldom are papillary tumors associated with calculous disease of the kidney

Papillary tumors of the renal pelvis are of clinical interest because of their relative position to the ureter (FIG 387) The impor

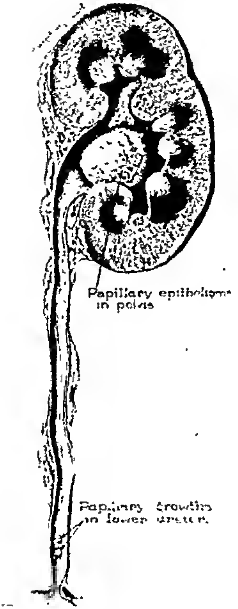


Fig. 387—Papillary tumor of renal pelvis.
(Courtesy of Dr. Dudley P. Fagerstrom and Journal of Urology, 1939, 41 137.)

tance of these tumors may be realized by the frequency with which the production of hydronephrosis occurs due to occlusion of the ureter and the frequency *with which implantation metastasis to* the ureter and bladder is observed. Implantation tumors in the

Fig 388—Squamous cell carcinoma of renal pelvis. Sagittal section of kidney revealing several large calculi filling nearly entire hydronephrotic renal pelvis Lower portion (which is upper pole), shows pyonephrotic cavities filled with growth The area about the hilus consists of a somewhat degenerated solid growth mass, also part of carcinoma.
(Courtesy of Dr Adolph A Kutzmann and *Journal of Urology* 1938, 39 487)

bladder may be single or multiple. These secondary tumors are usually observed as situated near the ureteral orifice of the affected side. Complete renal investigation should always be done in every instance where papillary tumor growths are observed in the blad-

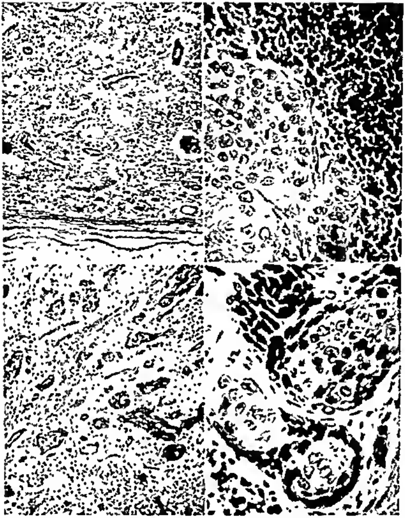


Fig. 389—Squamous cell carcinoma of renal pelvis.

Upper Left: Lowpower photomicrograph of an uninvaded portion of kidney revealing complete destruction with sclerosing of glomerular and tubular systems.

Upper Right: Highpower photomicrograph showing squamous cell carcinoma metastasis to a pedicle lymph gland.

Lower Left: Lowpower photomicrograph showing marked degree of invasion into kidney. Note complete absence of tubular system and there is one glomerulus.

Lower Right: Highpower photomicrograph of a group of cornified squamous cells forming an "epithelial pearl or whorl."

(Courtesy of Dr. Adolph A. Kutzmann and *Journal of Urology*, 1938, 39:487.)

der Beyond question the origin of many papillary vesical growths could be explained if such examinations were routinely done Likewise, it should be borne in mind that many instances of fatalities attributed to vesical malignancy would be shown to be directly due to the obstruction produced by papillary neoplasms rather than to the influences of vesical tumor growths

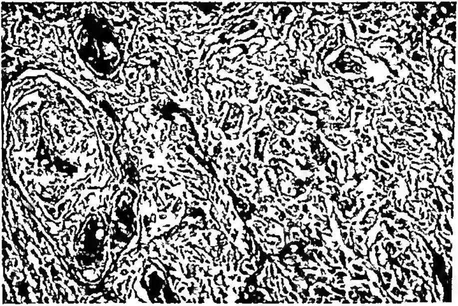


Fig 390—Kidney—Squamous cell carcinoma. Tumor cells show a considerable degree of differentiation with keratinization ("pearls") and the formation of intercellular bridges Stroma presents typical desmoplastic changes of the supportive connective tissue
(Courtesy of Dr Lawrence W Smith)

Squamous cell pelvic tumor is less frequently observed than the papillary type (Fig 388) Squamous cell tumors are highly malignant, metastasize widely and present a poor prognosis (Fig 389) There is a distinct relationship between squamous cell carcinoma of the renal pelvis, chronic infection, calculous disease and leukoplakia (Fig 390) The frequency of occurrence of the

various factors has not been fully explained. The squamous cell neoplasms may attain large size. They are of a grayish color, are flat and usually show extensive ulceration. They are indurated and show a definite tendency to infiltrate the pelvic wall with secondary involvement of the kidney (FIG. 391).

Symptoms: The cardinal triad of the symptoms of renal pelvic

Fig. 391—Low power photomicrograph. Typical acanthomatous type of squamous carcinoma of renal pelvis.
(Courtesy of Dr. Lawrence W. Smith.)

neoplasm is hematuria, pain, and tumor. These symptoms vary in degree of intensity and duration.

Profuse, irregular and intermittent hematuria occurs in from 70 to 80 per cent of the papillary type of renal pelvic growths and from 20 to 40 per cent of the flat, squamous cell growths.

Sudden profuse hemorrhage may produce a rapid increase in the size of the tumor in the loin. The sudden increase in size of the tumor may be accompanied by severe colicky pain with the

passage of blood clots down the ureter Bleeding may be present without pain or the presence of palpable tumor Pain may be absent or it may be intermittent The pain may be of a colicky nature due to the passage of clots through the ureter, or of a constant, stretching, burning fullness in the loin

Tumor is present in about 25 per cent of the cases It is usually due to the presence of hydronephrosis Seldom is a palpable tumor mass present with this type of neoplasm

Diagnosis· The preoperative diagnosis of renal pelvic tumor is comparatively difficult It is commonly confused with a diagnosis of parenchymal tumors There is no known clinical method of differentiation between papillary and squamous cell carcinoma Rarely is an early diagnosis of intrapelvic tumors made except in the instances that portray early hematuria and are subjected to complete examination to ascertain the source of the hematuria

Roentgenological examination may reveal the presence of a calculus which is relatively common in the squamous cell type Rarely do calculi produce profuse bleeding except in the presence of tumor growth Pyelographic studies usually reveal an irregular, space taking lesion when the intrapelvic tumor is of the papillary type

Cystoscopy Cystoscopic examination should be done if possible during the time of bleeding The side from which the bleeding occurs may be definitely ascertained If bloody urine is ejected from the ureteral orifice in the presence of vesical neoplasms, a primary tumor of the renal pelvis is strongly suggested It is estimated that vesical implantations occur in relatively 75 per cent of instances of intrapelvic tumors Severe ureteral bleeding following the introduction of a catheter to the renal pelvis, should arouse suspicion of renal pelvic tumor or a primary tumor of the ureter There is nothing in the appearance of the bladder that is pathog

nomonic of the presence of intrapelvic renal tumors. There are several findings that are suggestive: (1) The ejection of blood or bloody urine from the ureteral orifice; (2) the appearance of blood immediately following ureteral catheterization; (3) a papillary tumor growth at or near a ureteral orifice. This is particularly true if blood is seen to be ejected from the ureteral orifice.

Pyelography: Roentgenological examination is the most accurate method of diagnosis of intrapelvic renal tumors. The affected kidney may be of normal size and contour, or enlarged, or irregular in outline. Retrograde pyelography is to be preferred to intravenous urography. Intravenous urography may not present the sharpness of detail of the renal pelvis and calices as does retrograde pyelography. A space-taking lesion of small proportions may not be clearly outlined and may be overlooked more readily than when retrograde pyelography is used. Retrograde pyelograms clearly demonstrate any filling defects or irregular distortions of the renal pelvis. In some instances, dilatation or obliteration of some of the calices may be present. Small, neoplastic growths may not be of sufficient size to cause any alteration of the pyelographic shadow. It may be necessary to repeat the pyelographic studies at intervals in instances of unexplained recurrent hematuria. Hydronephrosis of varying degrees may be present depending on the presence or absence of obstruction of the ureter by the neoplasm. It should be borne in mind that a pyelogram made immediately after renal hematuria from any cause may be suggestive of intrapelvic neoplasm due to blood clots that may still remain in the renal pelvis.

Cystologic examination of the urine: Cystologic examination of the urine is more significant in tumors of the renal pelvis than in parenchymal tumors. Tumor cells and tumor fragments have been demonstrated in ureteral urine specimens. Villi and shreds

of leukoplakial membrane have been observed following ureteral catheterization. If such tumor cells are obtained during ureteral catheterization, their presence may be regarded as highly important diagnostic data.

Treatment: Treatment of intrapelvic renal tumors is nephrectomy. Nephrectomy should be done as early as a diagnosis may be made. It has been demonstrated on numerous occasions when the tumor has been treated locally or excised, that recurrence of the tumor is usual and that the recurrent tumor is more malignant than the original tumor.

3. *Hypernephroma*

Solid tumors of the kidney, occurring in adults, arise in the majority of instances in the parenchyma. Of these malignant neoplasms, hypernephroma, or Grawitz's tumor, is considered to account for 68 to 85 per cent of all renal tumors (PLATE LXXVI) Smith believes that renal adenocarcinoma (malignant nephroma) is the common primary tumor of the renal parenchyma, while the true hypernephroma of adrenal rest origin is a comparatively rare tumor of the kidney. The origin of hypernephroma has been and is the subject of great controversy since Grawitz, in 1883, suggested that these encapsulated renal tumors were probably of adrenal cell "rest" origin Such a theory has been opposed by Hinman, Ewing, Mackenzie and others. Ewing concluded that . . . "Sufficient evidence has been adduced to prove that the group of adrenal tumors of the kidney differs, as a whole, quite distinctly from renal adenocarcinoma with clear cells. The main destructive features of the adrenal growths are the central fibrous core and exact reproduction of the adrenal by benign growths and the general mesoblastic tendencies of the malignant forms. Chromaffin cells are not infrequently present. For an alveolar or papillary structure it

PLATE LXXVI—Hypernephroma. Natural color photograph.

is extremely difficult to establish an adrenal origin in man Yet Bothe presented a microphotograph that showed a hypernephroma in contiguity with a nodule of aberrant adrenal tissue thus supporting Grawitz s theory However, in refutation of such theory of origin a hypernephroma may show a tubular formation

Fig 392—Hypernephroma gross specimen, showing relative size of tumor mass

which is never demonstrated in the adrenal or in tumors of the adrenal cortex Tumors of the adrenal cortex are associated with sex disturbances and abnormal changes of the genitalia Such changes are never present in hypernephroma It is reasonable to assume that if hypernephroma does arise from adrenal cell rests such sex disturbances and abnormal genitalia should exist when hypernephroma is present It is inconceivable that such body changes taking place in true adrenal cortical tumors would fail to

show similar changes merely because of an aberrant position within a kidney.

Characteristics: Hypernephroma occurs with almost equal frequency in the upper and lower poles of the kidney. When the tumor occurs in the upper pole, invasion of the diaphragm and

Fig. 393—Hypernephroma. Retrograde pyelogram showing elongation and destruction of renal pelvis. Note position of the ureter.
(Philadelphia General Hospital.)

adjacent structures is common (FIG. 392). Nephrectomy may be an exceedingly difficult procedure. In size, hypernephroma may vary from well encapsulated tumors of three to four centimeters in diameter to enormous, irregular, cystic masses that fill half or more of the abdominal cavity. Hypernephroma occurs most frequently on the right side (FIG. 393). It occurs most commonly

between the ages of 40 and 60 with an average age incidence of 55 years Males are more frequently affected than females as 60 to 70 per cent occur in the male sex (Fig 394) The tumor has a great predilection for extending into the lumen of the renal vein

Fig 394—Hypernephroma. Retrograde pyelogram showing distortion and displacement of renal pelvis. Because of the marked gastrointestinal symptoms a gastrointestinal series had been previously done

(Temple University Hospital Acc No 45951)

and vena cava For that reason it metastasizes relatively early Metastasis is most frequently to the lungs liver brain and bony skeleton The metastatic lesions may be the ones to which primary diagnostic attention is focused It may not be until surgical pro

cedures for other reasons are done, or not until autopsy that the primary growth in the kidney is discovered. The size of the original growth does not influence the position or extent of the metastatic lesions (Fig. 395).

Fig. 395—Hypernephroma. Retrograde pyelogram showing marked distortion *and displacement of renal pelvis and calices.*
(Philadelphia General Hospital)

On section, the surface of the tumor presents a variegated appearance. Characteristically, the tumor is yellow (due to lipoid) interspaced with red hemorrhagic areas and cysts of varying sizes (Fig. 396). Some of the cysts contain serous or mucinous fluid. In

other cysts, there are areas of necrosis with spurious cyst formation into which hemorrhage has occurred Strands of connective tissue, which are occasionally calcific, may penetrate certain tumors

Fig. 396—Hypernephroma. Sagittal section Almost the entire kidney substance is replaced by the tumor growth
(Temple University Hospital Acc No 29 8)

Microscopically, the picture presented is a varied one, even in the same tumor Three major types of hypernephroma are seen The first type is one in which a tumor presents the typical picture of true adrenal cortex consisting of sheets and cords of pale, stain

ing, uniformly sized varicolated cells, with no suggestion of lumen formation (FIG. 397). The second type presents a very striking papillary adenocarcinomatous appearance and is made up of deeply staining granular cells with small hyperchromatic nuclei.

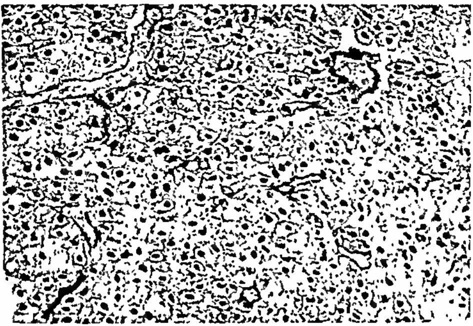


Fig. 397—Kidney—Hypernephroma, adrenal cell type. Tumor composed of sheets of relatively undifferentiated epithelial cells with small, ovoid, hyperchromatic nuclei and finely vacuolated cytoplasm; no acinar arrangement; delicate capillary stroma.

(Courtesy of Dr. Lawrence W. Smith.)

The cells are arranged on rather heavy, fibrous stalks (FIG. 398). The third type presents a tumor composed of so-called clear cells in which there is a definite attempt to form acini. These cells are large and coarsely varicolated, so that their cystoplasm is scarcely demonstrable except for the limiting membrane. The nuclei are large and round with well defined nucleolus and coarse rods and granules of chromatin. As a whole, these tumors are well vascu-

lirized, showing varying degrees of hemorrhage and necrosis (Fig 399)

Symptoms The cardinal triad of symptoms of hypernephroma is hematuria pain and tumor These three symptoms occur in combination in relatively 50 per cent of the cases In many in

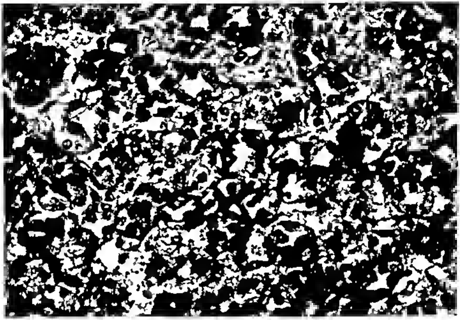


Fig. 398—Granular cells—Hypernephroma. Kidney—hypernephroma granular cell, renal type Tumor made up of small deep staining cells with well preserved nuclear detail Fatty vacuolization and granular degeneration are present. Note especially striking tendency toward lumen formation.
(Courtesy of Dr Lawrence W Smith)

stances one symptom is outstanding and overshadows the other two

Hematuria is by far the most constant symptom of hypernephroma Hematuria occurs in from 40 to 80 per cent of the cases according to various observers Hematuria may be a late symptom and may not occur until long after the development of the tumor Hematuria occurs only after the tumor has broken through

its capsule and invaded the renal pelvis. The bleeding is usually intermittent, moderate to profuse in amount, rarely is it fatal. Following the initial hematuria which is usually of a few days' duration, months may elapse before recurrence. Hematuria may be associated with pain in the loin or may simulate renal colic.

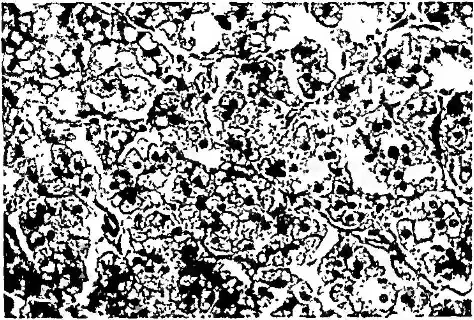


Fig. 399—Kidney—Hypernephroma, intermediate type. Adrenal-like, foamy, vacuolated cells arranged in cords (cf. zona fasciculata of adrenal gland); hyperchromatic nuclei, and finely vacuolated cytoplasm; no acinar arrangement, delicate capillary stroma.
(Courtesy of Dr Lawrence W Smith)

The colicky pain is due to the passage of clots along the ureter. Usually the blood is well admixed with urine and is without clot formation.

Pain: Pain is the second most constant symptom. The character, duration, and distribution of the pain may vary in every instance. The pain may be described as discomfort in the loin. It may be of the nature of an acute renal colic due to calculus. The

pain may suddenly occur and may be continuous, dull and aching in character This latter type of pain is caused by the tumor invading the surrounding structures It may be due to stretching of the renal capsule by hemorrhage within the tumor Pain in the groin or thigh is due to pressure on the lumbar nerves

Tumor The presence of a palpable tumor is the most infrequent initial symptom However, palpable renal masses are evident in 60 to 80 per cent of cases as the tumor enlarges These renal masses may be large or small, soft or hard, movable or fixed It is obvious that when such a tumor is so large that the patient is conscious of its presence, the chances of surgical removal are slight

Other symptoms Malaise, anemia, loss of weight, varicosities of the superficial veins of the abdominal wall, general cachexia are not uncommon symptoms of hypernephroma A mild, fibrile reaction not infrequently occurs and is thought by Wright to result from a foreign protein reaction to tumor particles in the blood stream Symptomatic varicocele may occur suddenly in males as the result of venous obstruction Symptoms referable to a metastatic growth may be the first clue to presence of the primary renal neoplasm

Diagnosis. Obviously, the success of treatment can only be *assured by early diagnosis Unfortunately, these cases come to* complete urological examination rather late This is due to the fact that such neoplasms are so insidious in onset There are no pathognomonic symptoms of hypernephroma Although the cardinal triad of symptoms hematuria, pain in the loin, and tumor are suggestive, the history of painless hematuria, intermittent in nature, pain, constant, dull, and aching in character, and the presence of a tumor mass are highly important factors but are not conclusive A history of hematuria of short duration, or a history of dull pain in the loin are not sufficient for a diagnosis Weeks of

constant observation and repeated urological study may be necessary before operative intervention is instituted. Of all the diagnostic procedures, the data made available by cystoscopy or cystoscopic procedures are of more significance than any other method.

Cystoscopy: There is nothing pathognomonic in the cystoscopic appearance of hypernephroma. The ejection of bloody urine from the ureteral orifice will disclose the affected side if examination is made at the time of bleeding. General examination of the bladder is negative. The presence of secondary papillary growths in the vicinity of the ureteral orifice, as is seen in papillary tumors of the renal pelvis, will be lacking. The ureteral orifice may show considerable variation in the rhythm of contraction. A ureteral orifice which is acting sluggishly should be regarded with suspicion of upper urinary tract disease, especially if the opposite ureteral orifice is acting normally. Even though the urine is clear and the appearance time of indigo-carmine is relatively normal, long intervals between ejection of the dyestuff and feeble movements of the ureteral orifice are suggestive of kidney damage. Varicosity of the veins surrounding the ureteral orifice is also very suggestive of renal tumor. The ureters are patulous unless compressed by the tumor mass. Ureteral catheterization is usually accomplished without resistance to the forward advancement of the catheter.

Renal function tests (indigo-carmine and phenolsulfonphthalein) are not very conclusive. On occasion, the affected kidney will show diminished function. This apparent normal function is due to the presence of sufficient secreting renal tissues to permit a relatively normal concentration and elimination of the dye. In the advanced stages of the disease there is usually a marked loss of function on the affected side.

Pyelography: Intravenous urography has not proven too effi-

cient in demonstrating minor calyceal deformities and distortion Retrograde pyelography is by far the most efficient in the demonstration of minor calyceal irregularities Retrograde pyelography is considered the most valued procedure in the diagnosis of renal neoplasm In more than 90 per cent of cases, distortion of the renal pelvis or calices occurs in the presence of renal tumor It is only after proper filling of the pelvis and calices that accurate interpretation of the x ray films may be made Distortion of the renal pelvis, as produced by a tumor growth, is caused by intrarenal pressure or the invasion of the pelvic cavity The pyelographic picture produced is most frequently one of distortion of the superior or inferior calyx The extent of this distortion varies so greatly that it is impossible to describe clearly all the variations

Treatment. The treatment of hypernephroma is that of early extraperitoneal nephrectomy if any success of cure is to be attained In the later stages of the disease, many factors must be considered before operative measures may be instituted The size and fixation of the tumor, the site and extent of metastasis, are salient points to be considered

4 *Adenocarcinoma*

(malignant nephroma)

Papillary adenocarcinoma of the kidney is a tumor affecting the renal parenchyma Ewing states that there is a clear cell and a granular cell variety The clear celled variety is a more or less solid tumor and is very vascular, exhibiting a marked tendency to hemorrhage, necrosis and cystic softening The clear cell type of malignant nephroma is usually a large single, invasive tumor (Fig 400) These tumors are occasionally encapsulated, but usually they are not These tumors are usually limited by the renal cap

sule and do not invade the perirenal tissue until the later stages. The clear cell variety of tumor shows a tendency to invasion of the renal vein with subsequent widespread blood-borne metastasis. The granular cell variety is less vascular and shows a tendency to metastasize by the lymphatics.

A third variety, alveolar adenocarcinoma, is also described.

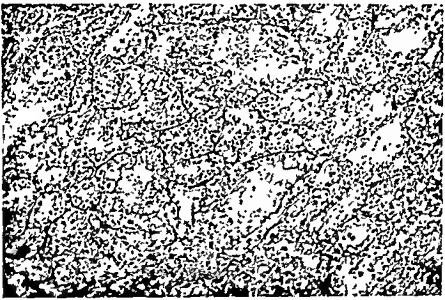


Fig. 400—Clear cell type of papillary adenocarcinoma, presumably of renal, tubular, epithelial origin.
(Courtesy of Dr Lawrence W Smith)

Alveolar adenocarcinomata are whitish, hematogenous tumors that infiltrate widely. In the early stage of the tumor, there is a distinctly invasive quality shown by the tendency to break through the renal capsule with infiltration of the perirenal tissue or by invasion of the renal pelvis. Microscopically, alveolar carcinoma show less cellular differentiation than any other type of renal adenocarcinoma. The cells are arranged in alveolar or tubular fashion.

Microscopically, it is not uncommon in adenocarcinoma that there is present an admixture or combination of the cellular elements which may present a confusing picture (FIG 401). Interspaced between remnants of normal renal cortex, cellular masses of the tumor may be found. These tumor cells may be arranged in a papillary, alveolar, or tubular arrangement. In other in-

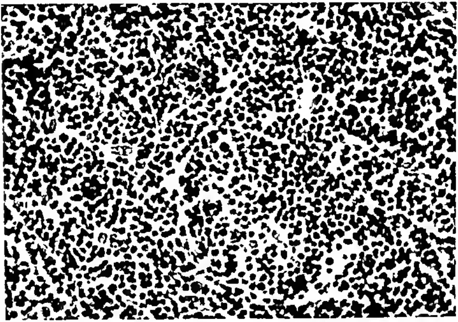


Fig. 401—Kidney embryoma (undifferentiated cell carcinoma) Kidney almost entirely replaced by a pearly white tumor made up of cells having no apparent architectural arrangement.
(Courtesy of Dr Lawrence W Smith.)

stances, the picture presented may so resemble hypernephroma that differentiation may be difficult (FIG 402).

It is usual that adenocarcinoma is well advanced when first seen clinically An enlarged, firm kidney is tightly bound to all the surrounding structures The invasion or attachment to the surrounding organs is due to the infiltrative characteristics and tendency of this type of tumor to become fixed An enlarged, palpable

kidney which exhibits fixation usually signifies inoperability (FIG. 403).

Symptoms: The two most frequently observed symptoms are those of hematuria and pain. The tumor occurs most frequently in men past 40 years of age. The right kidney is more frequently involved than the left. The condition is nearly always fatal either

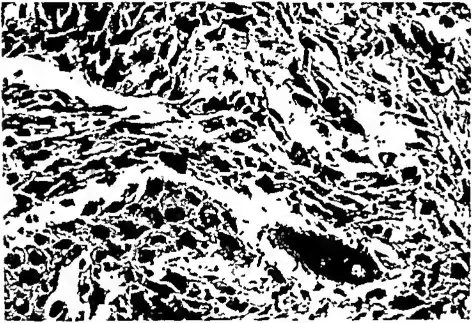


Fig. 402—Kidney embryoma (carcinosarcoma). An unusual tumor presenting evidence of both squamous carcinoma and diffuse fibrosarcoma.
(Courtesy of Dr Lawrence W Smith.)

as the result of the early, widespread metastasis, which is so characteristic of this type of tumor, or from recurrence of the tumor growth at the original site.

Diagnosis: The diagnosis of early renal adenocarcinoma may be exceedingly difficult if not impossible.

Cystoscopy: There is nothing pathognomonic in the appearance of the bladder that is suggestive of the presence of renal

adenocarcinoma There is little functional impairment of the kidney

Pyelography The pyelogram may reveal nothing that is suggestive of the presence of tumor Repeated pyelograms if the

Fig 403—Adenocarcinoma—Retrograde pyelogram showing a filling defect The lower and middle calices are so extended as partially to encircle the tumor mass
(Philadelphia General Hospital)

tumor involves the pelvis may show an obliteration of the nearest calyx Later as the pelvis is encroached upon several calices may be distorted or obliterated The renal pelvis may show marked distortion (Fig 404) Coincident with this obliteration of the pelvis and calices dilatation elongation, or retraction of the other

calices occurs. These elongated calices appear as very narrow, thin, curved, radiating streaks, with the classical appearance of "spider deformity" so characteristic of adenocarcinoma (FIG. 405).

Treatment: The treatment of adenocarcinoma is one of early

Fig. 404—Adenocarcinoma—Retrograde pyelogram. The pelvis is filled but the calices have been so compressed that visualization is impossible.
(Temple University Hospital, Acc. No. 39585)

nephrectomy, providing the kidney is not firmly adherent and there is no evidence of metastasis. As a whole, the prognosis of renal adenocarcinoma is poor. This is evident because of the tendency of the tumor to early, widespread metastasis and to the invasion of the surrounding tissues and organs.

Fig. 405—Adenocarcinoma—Retrograde pyelogram showing marked elongation of renal pelvis and distortion of the calices.
(Philadelphia General Hospital)

II

BENIGN MESOTHELIAL TYPE OF RENAL TUMORS

1. Connective Tissue Origin

(a) *Fibroma*

Fibromata of the kidney are usually small, subcapsular, nodular tumors. They may be single or multiple in number, unilateral or bilateral in position. Fibromata are frequently found at autopsy, but they are rarely of clinical importance. They are of clinical importance when they attain a large size. There are no pathognomonic signs or symptoms, nor urographic characteristics to distinguish them (Fig. 406).

(b) *Lipoma*

Intrarenal lipomata are rare. Normally, renal parenchyma contains no fat, but occasionally small intrarenal lipomata have been found at autopsy. Intrarenal lipoma must be differentiated from extrarenal lipoma and replacement lipomatosis. Substitution or replacement lipomatosis is not a neoplasm but a replacement of the normal renal parenchyma by fat. The origin of lipomata is a controversial issue, but it is generally conceded that they are possibly the result of fetal inclusions of embryonal tissue cells of the fatty capsule, enclosed within the parenchyma of the kidney. Clinically, lipomata are of little importance.

(c) *Chondroma and* (d) *Osteoma*

Isolated instances of osteoma and chondroma have been reported. They are of such rare occurrence as to be insignificant clinically.

Fig. 406—Fibrolipoma of kidney. Kidney after removal. Tumor attached to *posterior surface.*
(Courtesy of Dr. Henry G. Bugbee and *Journal of Urology*, 1941, 46:1.)

Fig 407—Benign leiomyoma of kidney Photograph of section shows encapsulated tumor and a portion of well preserved renal tissue at its lower pole Surface of tumor is whorling in configuration and bears a striking resemblance to a uterine leiomyoma.

(Courtesy of Dr O T Bailey and Dr J H Harrison and *Journal of Urology* 1937, 38 509)

2. *Myoma*

(*a*) Leiomyoma: Muscle tissue tumors of the kidney are rare Those reported in the literature have usually been small in size. Occasionally, leiomyomata of such size as to produce symptoms has been reported (*Fig 407*) Being small, solid cortical tumors, they do not present symptoms and are of little importance clinically unless such a tumor increases so rapidly in size as to produce the

symptoms of pressure. Leiomyomata of the kidney are structurally similar to leiomyomata of the uterus (FIG. 408).

(*b*) Rhabdomyoma: Rhabdomyoma of the kidney is rare. It does not differ from smooth muscle tumors found elsewhere in the body.

3. *Angioma*

Angioma rarely involves the kidney (FIG. 409). It is of interest and clinical importance because of the profound hematuria that occurs when the tumor erodes into the renal pelvis. Such tumors usually occur singly but may be multiple. The tumor occurs as a small, circumscribed, spongy mass showing no tendency to infiltrate. Angioma may be situated at the apex of a papilla, in the medulla, or rarely in the cortex of the kidney. Grossly, such tumors appear as benign tumors in which the blood vessels constitute the principal character of the growth (FIG. 410).

Symptoms: Gross, unilateral, renal hematuria and colic from

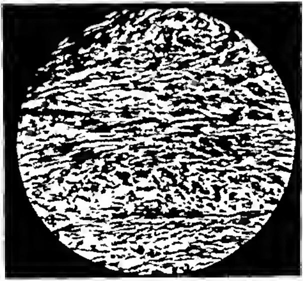


Fig. 408—Benign leiomyoma of kidney. Photomicrograph showing elongated smooth muscle cells with long, coarse, myogial fibers attached to cells.
(Courtesy of Dr. O. T. Bailey and Dr. J. H. Harrison and *Journal of Urology*, 1937, 38: 509.)

Fig 409—Hemangioma of kidney Sagittal section made through kidney and tumor mass
(Courtesy of Dr Harold H G le and *Surgery Gynecology and Obstetrics* 1939 48 555)

Fig. 410—Hemangioma of kidney Highpower photomicrograph showing spaces filled with red blood cells
(Courtesy of Dr Harold H G le and *Surgery Gynecology and Obstetrics* 1939 48 555)

the passage of clots are the most prominent symptom of angioma. The hemorrhages may be massive and brief, or moderate but continuous. In the latter instance, anemia may be extreme. There is nothing which will insure a diagnosis of angioma. Possibly the most important data may be gained by retrograde pyelography. Even with this procedure, there may be so little deformity of the renal pelvis or calices that a correct roentgenographic interpretation is impossible. Nephrectomy is the best procedure of treatment.

MALIGNANT TUMORS OF MESOTHELIAL ORIGIN

1. *Sarcoma*

Sarcoma of the kidney is relatively uncommon if the embryonal adenomyosarcoma seen in children are to be excluded. Sarcoma of

Fig. 411—Kidney—Sarcoma. Photomicrograph showing an extremely rapidly growing, highly undifferentiated *sarcoma*.
(Courtesy of Dr. Lawrence W. Smith.)

the kidney, in its true sense of structural formation, is very rare, although it is occasionally observed (FIG 411) Sarcoma is encountered in adults most frequently between the ages of 40 to 60 years. More frequently, the tumor is one of the mixed forms of tumor growth (PLATE LXXVII) Myosarcoma, liposarcoma (FIGS. 412,

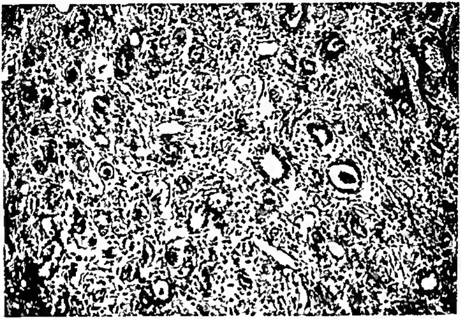


Fig 412—Kidney—Liposarcoma. Photomicrograph showing infiltration and destruction of renal parenchyma by fat cells in varying stages of differentiation (Courtesy of Dr Lawrence W Smith)

413), leiomyosarcoma, rhabdomyosarcoma are the types most frequently described in the literature. Fibrosarcoma of the kidney is rare.

There is nothing characteristic in the history or physical findings that would suggest the presence of sarcoma. The cardinal triad of symptoms observed in renal tumors is usually present. Hematuria, pain and tumor constitute the triad of symptoms. The distorted renal pelvis and calices, as revealed by pyelography, are the most diagnostic factors of the presence of a renal tumor.

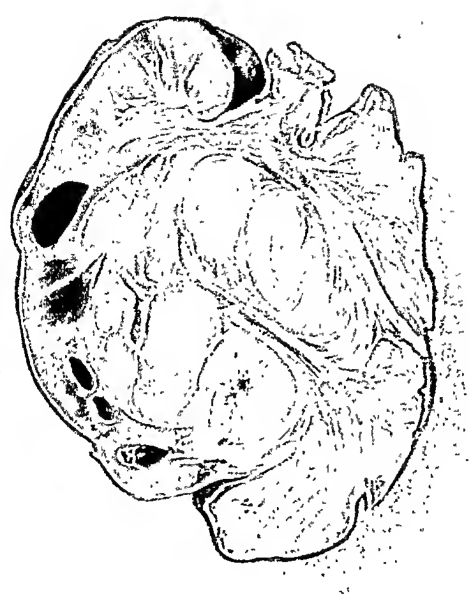


PLATE LXXVII—Liposarcoma of the kidney.
(Temple University Hospital, Acc No. 2828.)

Fibrosarcoma Large perirenal fibrosarcomata (retroperitoneal sarcomata) occur with relative frequency The condition occurs most frequently in men between the ages of 40 and 60 years The symptoms produced are those of palpable tumor and pain in the loin together with vague gastrointestinal symptoms The pain is

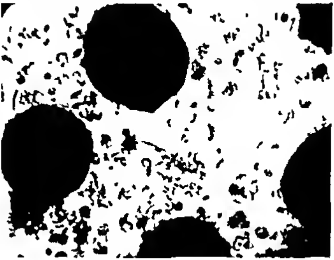


Fig 413—Liposarcoma of kidney Photomicrograph of tumor tissue stained with Sudan III

(Courtesy of Dr Charles Froug and *Journal of Urology* 1941 45 90)

usually dull and aching in character and results from involvement of the lumbosacral nerves Vague and obscure digestive symptoms associated with rapid loss of weight and strength are common Toxic symptoms such as fever, weakness and weight loss may be the first manifestations of the disease Upper abdominal distress food intolerance or vomiting may be the outstanding symptomatology Urine examinations are usually negative Symptoms referable to the kidney are usually absent although atrophic changes of the kidney are common The palpable tumor rather rapidly increases in size Pyelographic studies are occasionally negative but

usually show an obliteration of the renal shadow, pressure defects of the renal pelvis and displacement or rotation of the kidney. The ureter is displaced toward the midline of the body. Calcification within the tumor mass is not uncommon. Perirenal fibrosarcoma is a malignant disease with poor surgical prognosis.

Fig. 414—Kidney—Embryoma (rhabdomyosarcoma). Highpower photomicrograph from a malignant tumor of the kidney in a 10-year-old child. Tumor entirely made up of abortive, striated muscle fibers with a delicate connective tissue stroma.

(Courtesy of Dr. Lawrence W. Smith)

Removal of the tumor is usually followed by recurrence. X-ray therapy is of little or no benefit. The condition terminates fatally as a rule within one year after having first produced symptoms.

Rhabdomyosarcoma: Rhabdomyosarcoma of the kidney is a large, soft, grayish-pink tumor showing areas of necrosis and hemorrhage (Fig. 414). These tumors appear to be pseudoencap-

sulated but definite invasion of the renal tissue by the tumor may be readily seen The symptoms produced are not unlike those of other tumors of the renal parenchyma

III

MIXED OR TERATOID TUMORS

There are no benign types of mixed or teratoid tumors

Wilms' Tumor

(embryoma or adenomyosarcoma)

The commonest renal tumor found in infancy and childhood is embryonal adenomyosarcoma or Wilms tumor (PLATE LXXVIII) The tumor is highly malignant as is shown by the high mortality and infrequent cures This type of tumor is in reality an embryonal mixed growth In some instances the sarcomatous elements of the tumor predominate in others the muscle tissue elements are more prominent In other instances a variety of tissue elements is present This latter type presents a complicated histological picture Adenomyosarcomata are relatively rapidly growing tumors which attain considerable size The tumors are prone to be bilateral in distribution although this is not always true It may be difficult to ascertain whether both tumors are primary or whether one is a metastatic growth

With the possible exception of the eye the kidney is the most frequent site of malignant tumors in infancy and early childhood Adenomyosarcoma is the most frequent type of tumor of the kidney observed in infancy or childhood Duzan in an analysis of the regional distribution of 182 malignant tumors in infancy and childhood found the kidney or the eye involved in 115 instances However the tumor does not occur as commonly as these figures

PLATE LXXVIII—Wilms' tumor. Note the huge tumor mass almost completely obliterating the renal tissues.
(Temple University Hospital, Acc. No. 1923.)

would lead one to believe Bell found only 5 instances of adeno myosarcoma in 30 000 infant necropsies Dean and Pack found only 16 examples in the examination of 16,565 malignant tumors

Adenomyosarcoma is usually conceded to occur only in infants and young children Cases of this tumor have been reported as occurring in adults and even in individuals of advanced age The greatest incidence of occurrence is in the first five years of life Both sexes are equally affected Reference is made by some ob servers as to familial tendency

Pathogenesis The origin of adenomyosarcoma, as based on embryological interpretation, has been confused and controver sial Wilms, in his classical description of such tumors believed that they have a complicated etiology originating early in embry onal life from inclusion of fragments of the nephrotome (Wolffian body anlage) the sclerotome (primitive vertebral tissue) and the myotome (source of striated muscle) Such inclusions would ac count for the epithelial, mesothelial and myomatous elements found in these tumors There are many theories as to the origin of such tumors but none can fully explain their pathogenesis

Pathology The Wilms tumor is a large grayish white, encap sulated tumor of varying density, lying within a distended renal capsule The tumor may be solid, opaque and irregularly subdi vided into lobules It may contain cystic areas resulting from necrosis and present an appearance not unlike congenital cystic kidney

The tumor may originate from any part of the kidney, the upper pole, lower pole or midcortex They tend to remain encapsulated and sharply defined from the remaining portion of the kidney but once the capsule has been ruptured or penetrated by the tumor, growth may be rapid in all directions The kidney is rap idly destroyed as the result of compression or the kidney may be encircled by the tumor

The slow growth of such tumors is by expansion rather than by infiltration. For this reason, extensions and metastasis are exceptional in the early stages of the tumor growth. When metastasis does occur, the liver is the most frequent site of election. It is in the late stages that metastasis occurs to the lungs or skeletal struc-

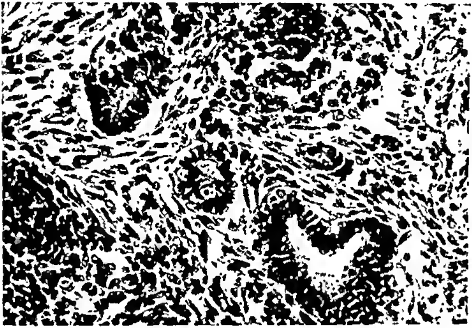


Fig. 415—Kidney—Wilms' embryoma (adenocarcinoma). Section shows characteristic mixed histopathology of the usual renal tumor of infancy and early childhood. Abortive glandular or tubular structure embedded in a complex connective tissue and smooth muscle stroma.
(Courtesy of Dr. Lawrence W. Smith.)

ture. There is a marked tendency of the tumor to recurrence following surgical removal.

Histologically, these tumors, composed as they are of such a variety of tissues, render a minute, detailed description impossible (FIG. 415). Ewing states . . . "The usual composition is of isolated tubules of high cylindrical or cortical cells with distinct lumina, surrounded by broad zones of indifferent spindle-cells, on

which is based the designation of adenosarcoma Either tubules or spindle-cells may be in excess, the tumor approaching embryonal adenocarcinoma or sarcoma "

Symptoms: The most frequently observed symptom is the presence of a tumor mass in the upper abdomen The size of the tumor mass varies in every instance In some instances, it is so large as almost to fill the abdomen Pain is usually a late symptom and results from pressure upon other organs In some instances weakness, fever, gastrointestinal disturbances, such as loss of appetite, constipation, or diarrhea may be associated with presence of the abdominal mass and pain

Hematuria is infrequent The absence of hematuria is attributed to the fact that there is usually no invasion or ulceration communicating with the renal pelvis by the tumor Loss of weight and anemia, either of which may be extreme, are usually observed late in the course of the disease Edema of the extremities, or ascites, may result from pressure of the tumor on the great veins Frequently, the superficial abdominal veins become enlarged and tortuous and varicose as the result of compression of the vena cava

Diagnosis. The diagnosis of Wilms' tumor is usually not difficult Abdominal examination should readily elicit the position, size consistency, and surface of the tumor Palpation usually reveals the tumor as a solid, smooth and rounded mass lying in the upper abdomen The tumor mass, being retroperitoneal, the intraabdominal organs are pushed forward, or to one side by the tumor

The plain roentgenogram seldom reveals any pertinent diagnostic data Cystoscopy and pyelography offer the greatest diagnostic aid available

Cystoscopy X rays of the abdomen, with a ureteral catheter *in situ*, will usually reveal a lateral displacement of the ureter Pyelography may reveal a normal appearing renal pelvis and

calices, even in the presence of a large tumor mass. This is due to the fact that there is little tendency for such tumors to invade the renal pelvis, but rather to enlarge at the periphery of the kidney. The pyelogram will usually show compression and elongation of the pelvis and calices. There is usually a lateral or downward displacement of the renal pelvis due to position of the tumor in relation to the kidney and the pressure exerted by it.

Individual renal function tests will show little or no difference in function in the early stages, but as the tumor enlarges and compression on the kidney increases, the affected kidney will show diminished function.

Treatment: Irradiation of the kidney, followed by nephrectomy, offers the greatest hope of cure. Nephrectomy should follow irradiation at a period no longer than three to six weeks, as these tumors rapidly become active after irradiation. The use of x-ray therapy alone, even though these tumors are markedly radiosensitive, has not proven satisfactory. The debatable question of the value of x-ray therapy preoperatively or postoperatively is a subject as yet undetermined, although many investigators believe preoperative irradiation is of benefit. Regardless of the type of treatment employed, none has given satisfactory results. The prognosis is always grave. Without treatment, death invariably ensues. When subjected to the most modern concepts of treatment, the mortality is exceedingly high, as few five-year cures have been reported.

XXIX

UROLOGY IN THE FEMALE

There will be no attempt to enumerate or discuss under this heading those urological conditions common to both male and female. Those urological conditions will be discussed that are incident to pregnancy, the puerperium and resulting from, or in association with diseases of the genital organs of the female (FIG. 416).

THE URETHRA

Until recent years, the female urethra has been one of the most grossly neglected organs in the human body. Being of short length and of relatively narrow caliber, its function as a conduit was considered sufficient until it was learned that the urethra of the female could be and was, on occasion, responsible for intense urinary symptoms.

The normal female urethra is a tubular structure. The *length* varies as given by different authors. According to Poirier and Charpey, the length of the urethra in the female is three centimeters; to Piersol, 3.5 centimeters; to Sappey, 3.8 centimeters; to Everett, 3 to 5.5 centimeters, with an average of 4.1 centimeters. The *lumen* of the normal adult female urethra is 26 F. The *meatus* is usually the most narrow portion of the canal. The urethra may be divided into three portions: The pelvic or posterior portion extending from the internal or vesical orifice of the urethra to the triangular ligament or urogenital trigone; the membranous portion lying between the two layers of triangular ligament and an external or vaginal portion that extends from the anterior leaf of the triangular ligament to the external meatus. The direction

taken by the urethra is not a straight line but rather that of a curve. Beginning at the vesical orifice, the urethra extends downward and curves somewhat forward to open at the external meatus, which lies in the midline of the vaginal vestibule about two centimeters posterior to the base of the clitoris.

The urethra is composed of three coats, the mucous, submucous, and muscular.

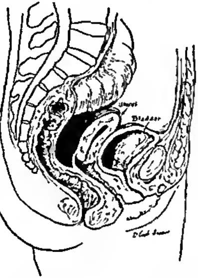


Fig. 416—Schematic drawing showing the normal arrangement and position of the pelvic organs in the female.

The mucous coat, comprising the epithelial lining of the external portion, is that of stratified columnar epithelium, but merges posteriorly as it nears the bladder into the transitional type of epithelium which normally lines the bladder, ureters, and renal pelvis. The muscular coat of the urethra is composed of three layers, an inner and an outer longitudinal layer and a circular layer. All these muscle layers are continuous with the musculature of the bladder. Kennedy considers that the involuntary sphincter is composed of the circular layer and considers it most efficient around the inner third of the urethra. The

voluntary sphincter is around the middle third and is derived from the urogenital trigone

Glands of the Urethra: There has been considerable controversy over the presence or absence of urethral glands There is no unanimity of opinion The presence of the compound racemose glands of Skene opening within the urethra is an established fact These glands lie in the submucosa of the posterior wall of the urethra and their ducts open on the floor of the urethra on either side of the median line The presence of glands other than Skene s is supported by MacKenzie and Beck, Hunner and Folsom, while Johnson, Cabot, and Shoemaker deny the existence of such structures Recently, Folsom again demonstrated that glands do occur and has resected portions of these glands that have become hypertrophied Microscopic examination of the tissue removed appears similar to hyperplastic prostatic tissue

Recently, Beneventi, in a study of 21 urethral specimens removed at autopsy, found the presence of glands in two instances that were indistinguishable from those of the infant male prostate

Diverticulum of the Urethra

Urethral diverticulum or urethrocele in the female is a pouch communicating with the urethra (PLATE LXXIX) It is formed by the dilatation of a portion of the urethrovaginal septum The origin of these diverticula is not known There is considerable dispute as to whether they are of congenital or acquired origin Many theories have been advanced as to the etiology of urethral diverticula (1) Some developmental defect, (2) the rupture of cysts in the urethrovaginal septum into the urethra, (3) trauma of the urethral walls during childbirth by a catheter or a calculus However, in the majority of instances a history of a long protracted, difficult labor is elicited

PLATE LXXIX—Cystoscopic appearance of the orifice of a urethral diverticulum in the female.

Symptoms and Diagnosis: The commonest symptoms presented are those of pain in the region of the urethra, dysuria, frequency, pyuria, pain on coitus, and the presence of a globulin tumor mass on the anterior vaginal wall. The pain in the region of the urethra and vagina is accentuated in the sitting position and during coitus.

Fig 417—Diverticulum of female urethra Cystogram shows a distinct globular shadow beneath the bladder
(Temple University Hospital, Acc No 7753)

There is usually a history of intermittent discharge of purulent urine from the urethra following digital pressure on the fluctuating *tender mass protruding into the vagina. The mass usually* increases in size during urination Hematuria is uncommon but may occur following an acute secondary inflammation or the formation of a calculus within the diverticulum.

The diagnosis of diverticulum of the urethra is not usually difficult. Examination reveals a fluctuating mass protruding into the

vagina. Compression results in the discharge of cloudy or purulent urine from the urethral meatus.

Cystoscopy reveals the presence of an irregular opening on the floor of the urethra.

Roentgenographic studies of the urethra reveal the presence of an abnormal pouch or sac lying below the bladder (Fig. 417).

Technic for x-ray demonstration: After filling the bladder with the opaque medium, the patient is asked to void while the urethral meatus is compressed. The opaque medium will readily enter the normal orifice, filling the diverticulous pouch.

Treatment: Treatment is that of total excision of the diverticulous sac through an incision in the anterior vaginal wall and a plastic repair of the urethrovaginal septum.

Acute Urethritis

Acute urethritis in women is, in most instances, due to an invasion by the gonococcus, though occasionally an acute inflammation may result from the invasion of other cocci or bacilli. Trauma or chemical irritation may predispose to a nonspecific inflammatory process. The causative organisms may usually be demonstrated and identified by bacterial cultures.

Symptoms and Diagnosis: Frequency, urgency, and a smarting, burning sensation on urination are the commonest symptoms. On examination, the urethral mucosa will appear red and edematous and appear to pout through the urethral meatus.

Treatment: The therapeutic measures are those necessary to reduce the concentration of the urine by increasing fluid intake. The urine should be rendered alkaline by the oral administration of alkalies such as sodium bicarbonate or potassium citrate. Warm sitz baths and the use of sedatives are beneficial to those patients suffering acutely. An attempt should be made to ascertain and eradicate the origin of the nonspecific infection as quickly as

possible following subsidence of the acute symptoms. These acute nonspecific inflammations tend to subside quickly if the exciting cause is removed.

Stricture of the Urethra

Stricture of the urethra is of more frequent occurrence than is generally conceded The caliber of the normal urethra in an adult female is 26 F. Any diminution of the urethral lumen smaller than 26 F is considered by many investigators as denoting the presence of stricture. The commonest site of such stricture is in the first two-thirds of the urethral canal.

The cause of stricture of the female urethra is not known although infection and trauma to the urethra during childbirth are important factors.

Symptoms and Diagnosis: The commonest symptoms are those of difficult or hesitant urination. The caliber of the urinary stream is lessened proportionately to the degree of narrowing of the canal. Acute urinary retention may occasionally be observed.

The diagnosis is made by the passing of bougies à boule of varying sizes The typical "hang" is readily elicited. Strictures of filiform caliber or those permitting the passage of a bougie 8 or 10 F are not infrequently seen.

Treatment: The treatment of urethral stricture is dilatation. Weekly dilatation with straight urethral sounds is recommended. The caliber of the sounds introduced is increased at each subsequent dilatation (Fig. 418).

URETHROCYSTITIS

Chronic urethrocystitis may cause women to complain of frequency of urination for years. The fact that the urine is positive on culture in many instances establishes beyond question that infection is basically the causative factor. Many of the conditions

productive of such a symptom have been previously described. To avoid unnecessary repetition, particularly in the section on cystitis, reference will be made, but a complete description will not be repeated. It is believed that in many instances the infection causing chronic urethrocystitis is extravesical in origin. Chronic, long-protracted cervicitis and vaginitis are undoubtedly responsible for a relatively high percentage of occurrence of the inflammatory changes that occur in the urethra or on the trigone. It is believed that the infection occurs not only because of the proximity of the external meatus to the vaginal canal, but also because of the penetration of the tissues by the offending organism from the

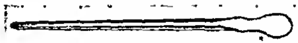


Fig. 418—Straight urethral sound. Such sounds are available in graduated sizes and are routinely used in the dilatation of the female urethra.

cervix uteri to the bladder mucosa. Repeated urine cultures will be negative in many instances, yet cystoscopically, definite pathological changes of the vesical mucosa will be observed. Many women may give a history of a cervicitis several years previous to examination and of occasional exacerbations of symptoms. A negative urine culture may be obtained, but cystoscopically these patients may show extensive changes of the vesical mucosa.

The pathological changes of the vesical mucosa are varied as to the area involved and their intensity. There are several conditions which are commonly observed and which it is believed are interrelated, progressing one after the other in successive steps of development.

1. *Granular Urethrocystitis*

It is believed that this granular appearing inflammatory reaction is the first stage, or at least the first visible evidence of a chronic

low grade infection of the vesical mucosa. Frequency of urination is the commonest symptom. There may be no other symptoms but the frequency may be so extreme that the individual may have an almost constant desire to void. In other instances, smarting and burning on urination may be acute and be associated with a frequent passage of only a few drops of urine.

The author, in 1940, reported an analysis of the examination of 500 women complaining of frequency of urination. A few presented a history of a previous gonorrheal vaginitis. The great majority did not. At the time of examination, all the women were or had been married. All the women examined had a cervicitis either at the time of examination or at some time previously. A culture of the vaginal discharge, when present, revealed the presence of *Bacillus coli*, staphylococci, nonvirulent streptococci, and diphtheroids These organisms were found either alone or in combination In many of the women examined, cultures of the bladder urine revealed negative results in repeated studies. It was impossible to ascertain the causative organisms producing such an inflammatory reaction of the vesical mucosa.

The persistence of urethrocystitis is suggestive of the co existence of a low grade chronic infection of the suburethral glands. The presence of such glands has been demonstrated by such men as Johnson, Sachs, Renner, Folsom, Caulk, Robin, and Cadit. The presence of such glands has been denied by such men as Cabot and Shoemaker However, it would seem feasible that such glands do exist and resemble the prostate in structure but in a less highly developed state. Once these glands become infected, a low grade infection of the urethra and trigone would continue for years. The etiology of chronic urethrocystitis is obscure The condition may be found at all ages but is commoner in the middle age of life in women who are married or have been married.

Urethrocystitis may involve the entire urethra and the trigone. It is observed most frequently in the posterior portion of the urethra and the adjacent portion of the trigone. In some instances, this granular appearing inflammation may surround the entire vesical neck; in others, it is localized on the trigone and extends into the urethra. Multiple localized granular areas may be occasionally observed. Normal appearing mucosa is interspaced between the granular areas.

Cystoscopy: These granular inflammatory changes do not have the appearance or the redness that is usually seen in acute inflammatory reactions. The changes in the mucous membrane appear as closely packed, small, fine, granular, slightly raised elevations. These elevations present an appearance not unlike coarse sandpaper. The flat, granular areas bleed readily with the slightest trauma. The granular appearance of the mucous membrane will not be seen if observed directly from above. The cystoscope must be turned so that the mucosa is seen at an angle to observe clearly the slightly elevated granular surface.

Treatment: Any treatment directed to the relief of the symptoms is usually followed with failure to cure. Urine culture is one of the principal guiding factors if a chronic infection of the bladder urine is revealed. Examination of the upper urinary tract, examination of the cervix uteri and eradication of any infection should be effected. A bland but nourishing diet should be outlined. Alcohol should be prohibited. Bowel elimination should be regulated.

Weekly urethral dilatation is highly beneficial. The dilatation of the urethra should be gentle and gradual. Straight urethral sounds should be used. Dilatation should begin with the largest sound that can be introduced without discomfort, usually size 18 or 20 F. The caliber of the sound should be increased by one number at each subsequent dilatation. Local application of silver

nitrate may give relief Copious bladder irrigation with 1 10 000 solution of silver nitrate may also be of benefit in serious cases

The oral administration of a urinary sedative may in many in stances control the frequency of urination and give considerable relief Alkalinization of the urine as an adjunct of treatment with such drugs as potassium citrate or sodium bicarbonate will reduce the smarting and burning of a highly acid urine The fol lowing sedative mixtures have given splendid results

If the symptoms are mild

℞ Potassium bromide		16 Gm	ʒ iv
Tincture hyoscyamus		16 cc	fʒ iv
Aqua camphorata	q s ad	120 cc	f℥ iv

Teaspoonful in water every four hours

If the symptoms are severe

℞ Potassium bromide		20 Gm	ʒ v
Potassium citrate		20 Gm	ʒ v
Tincture hyoscyamus		20 cc.	fʒ v
Tincture opii camphorata		30 cc.	f℥ i
Elix. sabol et santol	q s ad	120 cc	f℥ iv

Teaspoonful in water every four hours

It is believed that the use of an autogenous vaccine has been advantageous Vaccine therapy is considered valueless by many The use of vaccines is suggested when there is no infection of the upper urinary tract

2 *Cystic Formation at the Vesical Neck*

Polypoid or cystic formation at the vesical neck is believed to be a second stage or phase in the progressive reaction to long con tinued infection of urethrocystitis (PLATE LXXX) Three stages of development are believed to exist (1) Polypoid formation with out vascularization (2) polypoid formation with vascularization and (3) villous formation at the vesical neck The polypoid for

A

B

C

D

PLATE LXXX.

A. Polypoid formations of the vesical neck without vascularity.
B. Polypoid formations of the vesical neck showing distinct vascularity. Each cystic formation shows a distinct arterial supply.
C. Polypoid formations of the vesical neck. Grape-like appearance of one cystic area showing marked vascular supply.
D. Villous formation of the vesical neck. Each small villous projection shows a distinct arterial supply.

mation without vascularization may occur relatively early after the onset of the granular urethrocystitis The two conditions are frequently observed simultaneously The polypoid formations are usually observed at the superior margin of the vesical neck but may, on occasion, be seen on the lateral margin of the vesical orifice These polypoid formations appear as thin translucent,

Fig 419—Actual photograph showing polypoid formations of the vesical neck in the female that are avascular

Fig 420—Actual photograph showing polypoid formations of the vesical neck in the female that are distinctly vascular

grayish cystic projections without apparent blood supply, hanging from the superior rim of the vesical neck (Fig 419) They may be seen as a single projection but they are usually multiple At a later time it is believed that these apparently unvascular polypoid formations undergo vascularity (Fig 420) The blood vessels are rather prominent and are capable of the production of hematuria (Fig 421) The villous type of formation presents an

entirely different appearance. The villous projections are considerably longer. The villi are relatively the same diameter from the base to the tip. They present a distinct shaggy fringe around the superior arc of the vesical neck. Each projecting villus has a large, single artery of sufficient size to cause marked hematuria if

Fig. 421—Actual photograph showing marked vascularity of polypoid formation.

Fig. 422—Actual photograph of solid appearing polypoid formation on the vesical neck.

ruptured. On two occasions, the author has seen such extensive *intravesical hemorrhage following rupture of the blood vessels* that immediate fulguration of the bleeding points was necessary. Both patients required blood transfusions to restore the great blood loss.

Treatment: The treatment of polypoid or cystic formations of the vesical neck is one of weekly urethral dilatations and irrigations with silver nitrate solution (Fig. 422). As the condition is

usually observed in women in middle life or past such an age, dilatation should never be carried to the extreme (Fig. 423). The greater the age of the patient, the more carefully should dilatations be increased above 26 F. The villous projections respond poorly to dilatation. They should be removed by the fulguration current or may be removed by the cutting loop of the McCarthy resectoscope. The urethra should be dilated at weekly intervals by straight urethral sounds following the removal of the villi.

Fig 423—Actual photograph of irregular polypoid mass on the vesical neck. Note normal appearing vesical mucosa in background

3. *Irregularity and Contractions of the Vesical Neck*

Irregularity and contractions of the vesical neck can hardly be classified under the heading of the previously discussed subjects, although they are believed to be the result of inflammation It has been our experience that in every instance where the condition was observed, the major symptom is difficulty in starting the urinary stream Slight dribbling occurs after urination in the majority of instances. The loss of a few drops of urine on sneezing or coughing frequently occurs. The age of women suffering with such irregularity and contraction is generally higher than the age of women having urethrocystitis.

Cystoscopy · Irregularity of the vesical neck presents a rather bizarre appearance (Plate lxxxi). The margin of the vesical

PLATE LXXXI—*Irregularity of the bladder neck. The mucosa of the vesical orifice appears stiff and irregular in contour.*

neck is irregular in contrast to its normal smooth contour The irregularity may assume a series of clefts or folds at one or more points around the vesical neck The clefts or folds are usually most marked just below the superior arc on the lateral margin of the vesical neck There is usually an associated narrowing of the vesical neck This narrowing or contracture of the vesical neck may be extreme even to a point of producing a sense of resistance as the cystoscope is introduced As observed the mucosa of the vesical neck appears stiff and rigid and is lighter in color than normally seen The stiff and rigid nature of the mucosa is particularly noted at the site of the irregularity

Treatment The treatment of irregularity of the vesical neck is gradual urethral dilatation The dilatations should be done at weekly intervals The caliber of the dilating sound should be increased by one size at each subsequent dilatation Due to the age of the patient and the definite rigidity of the vesical neck that is present it is advised that dilatation should never be carried above 28 F This condition portrays a definite tendency to recur and it may be necessary to repeat the dilatations at rather frequent intervals

4 *Pseudomembranous Cystitis*

Pseudomembranous cystitis is the result of a chronic inflammation and occurs only in the female bladder This pseudomembrane is most usually confined to the trigone The mucosa of the trigone is covered by a whitish semitransparent pseudomembranous exudate The surface and borders of the exudate are irregular and appear slightly elevated above the surrounding normal mucosa The condition may be observed within a bladder that otherwise appears normal and the urine is sterile on culture The symptoms produced by this lesion are those of frequency smarting and burning on urination The condition

is frequently observed in the presence of a cervicitis or in individuals who have had a nonspecific vaginal discharge.

This condition has been more completely discussed under the general section on cystitis.

5. *Cystitis Vetularum*
(cystitis of aged women)

Cystitis vetularum is a part of a generalized senile atrophic process. The condition is found only in elderly women. As the result of a nonelastic contracted bladder, urinary frequency and discomfort may be marked.

Treatment is unsatisfactory although some relief may be gained by bladder irrigations.

6. *Mosaic Cystitis*

Mosaic cystitis is a condition observed in elderly women (PLATE LXXXII). The mucosa appears as a regular pattern of mosaic design. It is from this appearance that the condition derives its name. There are no definite symptoms. The treatment is one of the routine treatment of cystitis.

7. *Prostatism in the Female*

The presence of glandular structures causing obstruction to the vesical outlet in the female has been reported by Folsom, Caulk, Nesbit, Fite, and Van Houtum. Folsom recently presented in detail several photomicrographs showing the resected tissue of the vesical orifice of a female which was, in appearance, histologically similar to the prostate in males. He states that clinically, besides bladder irritation, some degree of difficulty of urination was present. Pyuria was frequently observed. Three of 15 cases showed complete urinary retention. He states that . . . "On

cystoscopic examination, one may find a normal bladder wall or a grossly trabeculated wall with cellules or even diverticula and calculi. The best observation of the bladder neck, in our opinion, is made by using both the close vision cystourethroscope and the panendoscope. At times we have found a retrograde lens of great value in evaluating a collaret type of enlargement. Not infrequently the bar or collaret may be felt by palpating the urethra through the vagina with the cystoscopic sheath in place."

Treatment: Treatment, as suggested by Folsom, is that of trans urethral resection of the hyperplastic tissue.

THE BLADDER AS INFLUENCED BY THE UTERUS IN ITS PHYSIOLOGICAL AND PATHOLOGICAL STATES

Pregnancy: Cystoscopy during pregnancy is not an uncommon procedure. The major urologic problems during pregnancy are confined to the upper urinary tract. In some instances, ureteral catheterization is necessary for diagnosis and treatment. There are few contraindications to cystoscopy during pregnancy. There are some alterations of the bladder during pregnancy. These alterations may be considered as: (1) Intrinsic; (2) extrinsic.

1 The bladder, during pregnancy, shows nothing unusual except vascular engorgement. The mucosa is of a deeper color than normally seen in the nonpregnant woman, as all types of blood vessels are dilated. The small veins become greatly dilated and in some instances show pronounced varicosity. This hyperemia is observable over the entire mucosa, but is most marked over the trigone. The trigone is broadened between the ureteral orifices and is lengthened from the urethra by pressure exerted by the enlarged cervix.

2 The results of extrinsic pressure vary in degree with the stage of the pregnancy. This is evidenced by the forward bulging

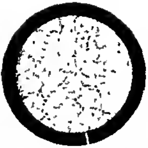


PLATE LXXXII—Mosaic cystitis. The vascular pattern presents a mosaic design.

of the posterior wall of the bladder as the enlarging uterus exerts its ever increasing pressure. No difficulty is experienced in doing cystoscopy until the fifth month of pregnancy. At that time, there is a diminution of the anteroposterior measurement of the bladder, which measurement decreases as the pregnancy advances to term. During the last month of pregnancy, the presenting part of the fetus causes additional pressure. The posterior bladder

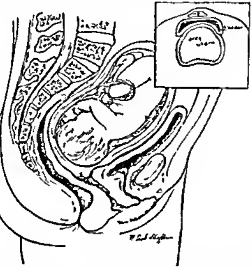


Fig 424—Schematic drawing showing the displacement of the bladder by the gravid uterus Insert illustrates the relative distortion of the bladder by the gravid uterus at term.

wall becomes convex instead of concave and causes the bladder to assume a crescentic or half-moon shape (Fig. 424)

The bladder remains atonic during the puerperium. During this period of atony, residual urine and urinary retention are of common occurrence A return to the normal gradually takes place, first with the return to normal micturition, and then with a complete loss of the residual urine.

The gravid uterus may present difficulties in the introduction of the cystoscope that are not encountered at any other time. These

difficulties increase as the pregnancy approaches term. The technic of the introduction of the cystoscope is more fully discussed under Pyelonephritis of Pregnancy.

Uterine Displacements: Uterine displacements will, according to the type and degree of displacement, produce definite alterations in the contour of a bladder that is otherwise normal.

Under normal conditions, the cervix lies at the level of the bas-fond and upper limits of the intraureteral ridge. The cervix does not interfere with the normal contour of the base of the bladder. The fundus of the uterus lies immediately behind the posterior wall of the bladder and normally forms a slight boss or indentation on the posterosuperior wall. The cystoscopic distending medium is sufficient to cause the bladder to assume its rounded contour without indentation as the bladder is filled.

Anteflexion: Anteflexion is an exaggeration of the normal position of the uterus in relation to the bladder. If marked, anteflexion will increase the boss formed by the uterine fundus and tend to lessen the measurement between the posterosuperior and the anterior walls of the bladder. The distending medium is sufficient, unless the condition is extreme, to erase the indentation and cause the bladder to assume its normal contour.

Retroversion and Retroflexion: The uterus in retroversion or retroflexion is bent backward toward the rectum causing the cervix to be pushed upward against the vesical wall in the region of the bas-fond and intraureteral ridge (Fig. 425). The vesical floor and trigone are elevated with the production of a deepening of the lateral recesses of the bladder. The ureteral ridge is rotated outward, causing the orifices to open on the lateral surface instead of opening on the superior surface. The dome of the bladder is falsely thrown forward, creating a greater distance from the dome to the vesical orifice than is normal.

Cystocele Cystocele is a downward protrusion of the bladder and anterior vaginal wall Cystocele is always associated with some degree of uterine prolapse In the presence of procidentia

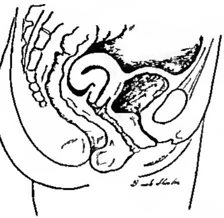


Fig. 425—Effect on the bladder in the presence of retroflexion of the uterus.

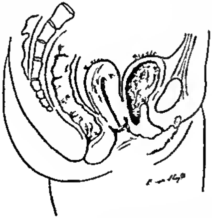


Fig 426—Schematic drawing illustrating the presence of a moderate cystocele due to uterine prolapse

the cystocele is the greatest part of the protruding mass from the vaginal orifice The condition usually follows childbirth but it may rarely occur in nulliparous women or even in virgins (Fig 426)

Cystocele usually results from the laceration of the muscle of

the urogenital trigone, the diastasis of the anterior vaginal fascia and the elongation of the ureterovesical and cardinal ligaments.

Cystocele may be slight or may be so extensive as to permit the greater portion of the bladder to protrude through the vaginal orifice. The symptoms produced are mainly those of vesical irritation except for the protrusion from the vulva. The bladder symptoms are those of frequency and urgency of urination resulting from the decomposition of the residual urine with secondary inflammation of the vesical mucosa (Fig. 427).

Fig. 427—Schematic drawing illustrating advanced cystocele in the presence of uterine prolapse.

Cystoscopy may be difficult in the presence of a cystocele. This difficulty is due to the downward and forward direction which the trigone and the floor of the bladder has assumed. The floor of the bladder may recede very acutely. Introduction of the cystoscope may be accomplished in extreme cases by elevating the hilt of the instrument above the pubis. On occasion, visualization of the trigone is accomplished only by the elevation of the ocular end of the cystoscope in a vertical plane instead of the horizontal plane that is customary. It is possible temporarily to restore the bladder to a relatively normal position before cystoscopy by the

insertion of a vaginal tampon Visualization of the entire bladder is permitted by means of this procedure If the floor of the bladder is not elevated, the trigone may only be observed by awkward, wide excursions of the cystoscope The trigone, in the presence of cystocele, forms the anterior wall of a redundant pouch Being in such a position, catheterization of the ureters may be impossible In the presence of a cystocele, complete vesical distention is imperative as the anterior and posterior walls of the bladder will lie in close apposition On observation, the apposition of the walls will appear as a deep crease running transversely across the bladder This crease will be observed to widen and then disappear as the bladder is filled with the distending medium

Observation of the bladder will reveal trabeculations The intraureteral ridge will appear hypertrophied and the ureteral orifices will appear more widely separated than normal If infection is present, the mucosa will be inflamed in varying degrees of intensity

Treatment: The treatment of cystocele does not routinely come within the scope of urology but is rather one of gynecology, although the symptoms from which these women complain are mainly those of vesical irritation The treatment depends on many factors such as age, the extent of the cystocele and symptoms produced The treatment may be palliative or radical The palliative method of treatment is one of the institution of a pessary in one of its many types The radical treatment is that of the plastic repair of the vesicovaginal septum

Tumors of the Uterus Tumors of the uterus, either benign or malignant, affect the bladder in one way or another as the uterine pathology is increased

Fibroid tumors of the uterus (Fibromyomata uteri) Fibroid tumors of the uterus affect the shape and contour of the bladder as the result of extrinsic pressure Symptoms referable to the bladder

depend upon the position, number, and size of these fibromyomatous tumors. Relatively 50 per cent of women suffer with some degree of uterine fibroid formation. Most of these uterine fibromyomata remain insignificant. They do not cause symptoms and do not require treatment. Fibroid tumors are rare in women under 25 years of age. Fibromyomata usually begin their growth between the ages of 25 and 40. New tumors rarely develop after the menopause and rapid growth of a tumor at this time of life is highly suggestive of sarcoma.

There are four types of uterine fibroids classified as to their position: (1) Interstitial or intramural, when the fibroid lies in the uterine wall; (2) subserous, when the growth of the fibromyoma is directed outward toward the peritoneum; (3) submucous, when it bulges into the uterine cavity and; (4) intraligamentary, when the tumor has grown between the layers of the broad ligament. Those uterine fibromyomata that affect the bladder in the greatest number of instances are the interstitial or intramural fibroids that occur in the anterior wall of the uterus. It is in this position that the fibroids are in close approximation with the bladder. The bladder is affected greatly by the relative position of the tumor as regards the longitudinal axis of the uterus. If the tumor lies in the anterior wall near the base of the uterus, pressure is directed toward the vesical outlet. Chronic urinary retention results. Associated vesical dilatation and hypertrophy and secondary infection are usual. The trigone and floor of the bladder are elevated. The ureteral orifices are wider apart than normal and open upon the outer surface of the ureteral ridge.

When a small tumor is situated in the body of the uterus, there are no symptoms or alterations in the normal cystoscopic picture. A large fibroid tumor of the body of the uterus not only causes a marked elevation of the floor of the bladder, but tends to compress the entire bladder against the symphysis pubis with more

or less complete obliteration of the vesical cavity. The cystoscopic picture presented is similar to that of the bladder in the latter stages of pregnancy. The anteroposterior diameter is reduced and the bladder extends laterally with the formation of deep lateral recesses Introduction of the cystoscope is only accomplished by advancing the *instrument obliquely into the lateral recess.*

Treatment is not one of urologic procedure but rather one of gynecological surgery.

Carcinoma of the Cervix and Uterus: The cervix is the commonest site of carcinoma in the human body Carcinoma of the cervix occurs about four times as frequently as does carcinoma of the body of the uterus It is commonest between the ages of 40 and 50, but is occasionally seen before 30 years of age and after 60. Considerable importance should be placed on the question of vesical involvement preoperatively The bladder should be explored as a routine measure in instances where a diagnosis of carcinoma of the cervix has been made. In the great majority of instances, the bladder will present a normal appearance. Definite pathology of the bladder will be frequently observed. The slightest evidence of invasion of the bladder by extension of the malignant growth should be considered as a contraindication for hysterectomy.

Cystoscopy The mechanical effects of obstruction of a malignant growth are, in general, considerably less than those observed in the presence of uterine *fibroids* It is a frequent cystoscopic finding to observe the floor of the bladder elevated to varying degrees. This elevation cannot be considered as definite evidence of infiltration Actual infiltration of the vesical wall and mucosa produces several definite and characteristic changes The bladder wall appears rigid and stiff Definite transverse ridges of the posterior wall are a frequent observation Such ridging is significant of fixation The earlier signs of invasion are those of dilatation of the blood vessels and minute petechial hemorrhages in the mucosa of the

area that has become infiltrated. Bullous edema may surround this circumscribed area or present a more extensive appearance across the trigone and floor of the bladder. As the infiltration continues, a definite yet sharply localized irregular nodule appears over the infiltrated area. This nodule tends to spread from its periphery. The surrounding area shows extensive bullous edema. In some instances, superficial yet localized necrosis of the infiltrating nodule occurs. Should this tissue necrosis continue, the formation of a fistulous tract between the bladder and cervix develops. The ureteral orifice or orifices are frequently involved in malignant extension from cervical carcinoma. Many of the fatal terminations of such malignancy do not directly result from the original growth but rather are the result of renal back pressure with subsequent hydronephrosis and ultimate uremia. The ureteral orifices may be so completely obscured by bullous edema as to render visualization or catheterization impossible. The orifices may, on occasion, present a gaping retracted appearance with such fixation that the orifices remain open at all times. In other instances, catheterization of the ureters for study or drainage is impossible due to stricture or scar tissue formation. As in all other forms of obstructive uropathy, dilatation of the urinary tract above the stricture is inevitable.

Intravenous urography will be of material aid when retrograde pyelography is impossible. Much can be learned from such roentgenological study.

Because of all these incipient yet definite pathological changes, every woman suffering with carcinoma of the cervix or body of the uterus should be given the benefit of complete urological study. These studies should include cystoscopy and urography (retrograde or intravenous).

Urogenital Fistula: Fistulous communication between the urinary and genital systems or tracts is not uncommon. The urinary

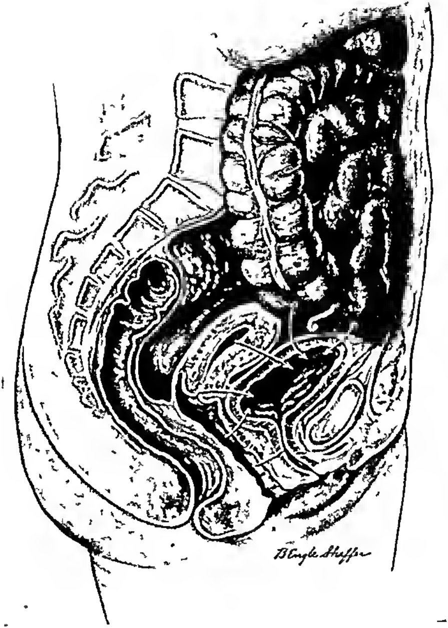


Fig. 428—Drawing illustrating the possible routes of vesical fistula in the female

dribbling resulting from such a fistulous tract is usually painless, constant as well as distressing. Such fistulous tracts usually result from:

(1) Obstetrical trauma caused by necrosis of the intervening tissues between the head of the infant and the bladder in instances of prolonged or difficult instrumental delivery.

(2) Postoperative sequel following pelvic operations, usually hysterectomy, either by the abdominal or vaginal route.

(3) The prolonged application of radium for treatment of carcinoma of the cervix.

Fig. 429—Actual photograph of the orifice of a vesicovaginal fistula.

(4) Carcinomatous extension from the cervix to the bladder.

The fistula may be classified according to the position (Fig. 428). Considerable study may be necessary to locate the orifice of such fistulous tracts. All fistulous tracts present one constant symptom, namely, dribbling of urine.

(1) *Vesicovaginal Fistula:* Vesicovaginal fistula implies an opening in the vesicovaginal septum. This is the commonest type of fistulous tract formation in the female. The orifice may be very high or at the level of the trigone (Fig. 429).

(2) Vesicocervicovaginal fistula implies a communication between the bladder and vagina at the junction of the cervix and vagina.

(3) Vesicouterine or intracervical fistula implies an opening from the bladder to the cervical canal so that urine has its exit through the external os.

(4) Urethrovaginal fistula implies a communication between the urethra and vagina.

(5) Ureterocervical fistula implies a communication between a ureter and the stump of the cervix so that the urine has its exit through the external os.

(6) Ureterovaginal fistula implies a communication between a ureter and the vagina.

Usually there is only a single orifice which may vary in size from one that is minute to one of large caliber. In fistulous tract formation, there is one symptom that is constant in all, namely, continuous urinary dribbling. The one condition from which differentiation must be made is that of ectopic ending ureter. The history of urinary disturbance only or since recent difficult delivery or operation is the establishing point of differentiation. In ectopic ending ureter, dribbling of urine would have been apparent since birth.

The clinical diagnosis of the type of fistulous tract may be difficult and tedious Repeated examinations may be necessary to locate the orifice Cystoscopy should always be done to locate the vesical orifice of the tract. In some instances, digital examination may reveal the location of the orifice, particularly of a large vesicovaginal fistula. The use of a colored fluid indicator is always of aid. The instillation of an aqueous solution of mercurochrome into the bladder, noting its escape into the vaginal canal is a safe procedure If red colored fluid escapes into the vagina, a vesico vaginal, vesicocervicovaginal or a vesicouterine fistula is present. If the fluid escaping into the vagina is clear, it is obvious that the communication is above the bladder. The intravenous adminis

tration of 5 cc. of a 0.4 per cent solution of indigo-carmine and the appearance of a blue colored fluid from the cervical os or vaginal fornix will establish the presence of a fistulous tract with the ureter. The obstruction to the passage of a ureteral catheter, in the presence of a fistula, is not sufficient evidence to warrant a diagnosis of postoperative ureteral fistula. Unless the orifice of the fistula is obvious, intravenous urography should always be done as a routine procedure.

Treatment: Successful correction can only be accomplished by the reëstablishment of the continuity of similar structures by plastic procedures.

Pyelonephritis of Pregnancy: Renal infections during pregnancy and the puerperium are common. The condition is frequently bilateral, or if unilateral, the right kidney is more often affected than the left. Renal infection may occur at any stage of pregnancy, but it usually occurs between the fourth and sixth months. Pyuria may exist without evidence of renal infection and the condition may be of such mild proportions as to go unrecognized unless repeated microscopic examinations of the urine are done. Initial pyelonephritis is more frequent in primiparae than in multiparae. Former normal pregnancies do not insure against pyelonephritis, nor is it necessarily true that subsequent pregnancies will be complicated by renal infection. However, it is possible and frequently does occur that pyelonephritis complicates each pregnancy. In multiparae, pyuria without actual symptoms is suggestive of a previous infection which may date back to a former pregnancy and which has existed sufficiently long enough to cause serious renal damage. In primiparae, the occurrence of pyuria is usually associated with the typical signs and symptoms of renal infection. The clinical picture of pyelonephritis of pregnancy does not differ from that of acute pyelonephritis of the nonpregnant female. The

course varies from a very mild pyuria to a severe acute diffuse pyelonephritis with all coincident signs and symptoms of massive renal infection

Etiology Stasis anywhere in the urinary system is definitely the most important predisposing factor in infection The dilatation of the renal pelvis and ureter incident to normal pregnancy is undoubtedly a great predisposing factor Lee and Mengert have shown that pregnancy causes a definite dilatation of the ureters, which is undoubtedly a great predisposing factor Lee and Mengert have also shown that pregnancy causes a definite dilatation of the ureters which is usually more marked on the right and is a normal concomitant of pregnancy ' Crabtree and Prather, Duncan and Seng and others have found some degree of dilatation in the upper urinary tract in all cases, the right showing dilatation in 100 per cent of the cases, the left, 70 to 85 per cent Lee and Mengert concluded that such a dilatation was to be considered a normal occurrence and that the dilatation subsides rapidly following termination of normal pregnancy and puerperium These investigators demonstrated a marked decrease in 24 hours To prove their contention that such a dilatation was a normal occurrence in pregnancy 15 normally pregnant women were subjected to ureteral catheter drainage for 24 hours without evidence of any change in the degree or character of the dilatation of the renal pelves or ureters Kretschmer, Heaney, and Ockuly studied 54 women between the second and fifth month of pregnancy and concluded that

1 Dilatation of the ureters and kidney pelves occurred in 100 per cent of our cases during pregnancy or the puerperium
2 The striking feature about the dilatation of the ureter during pregnancy is that dilatation is almost universally above the brim of the pelvis

3. As a rule, the dilatation is progressive along with pregnancy. There was one exception to this state, in which the ureter was normal in advanced pregnancy when earlier, it was dilated.
4. Lateral displacement of the ureter, when found early in pregnancy, tends to increase as the pregnancy advances.
5. In none of these cases did pyelitis develop during pregnancy, although marked dilatation and lateral displacement were present.
6. Presentation and position of the fetus could not be brought into causative relationship with dilatation or displacement."

These investigators studied 32 cases through the puerperium and found that 59.3 per cent returned to normal in two weeks; 34.3 per cent after six weeks; the remaining 6.2 per cent were normal after 12 weeks.

As the result of such work, there is sufficient evidence to conclude that dilatation and retention in the upper urinary tract are constant factors in pregnancy.

There are several theories as to the underlying factors in the production of this dilatation.

Mechanical: The obstructive or mechanical theory is one in which the ureteral dilatation and subsequent dilatation of the upper urinary tract are secondary to mechanical origin. Hofbauer is of the opinion that hypertrophy and hyperplasia of the muscle and fibrous tissues of the ureter and periureteral sheath are responsible for the obstruction. Schumacher is of the opinion that the dilatation is due to pressure on the ureter between the gravid uterus and the brim of the pelvis. However, Lee and Mengert found that in retrograde pyelography of ten pregnant women, no evidence of pressure on the ureter from adjacent structures could be elicited. They concluded that by draining the ureters with

ureteral catheters no change in the degree or character of the dilatation of the upper urinary tract was produced

Muscular atony Muscular atony of the ureteral and pelvic musculature resulting from the action of either hormones or toxins is advanced by some observers Each theory has its supporters but as yet no true hormone or toxin has been isolated that is capable of producing ureteral dilatation so universally observed

Undoubtedly many factors some of which are yet to be explained play a part in ureteral and pelvic dilatation during pregnancy Regardless of the causative factors dilatation of the ureters and renal pelves with coincident stasis definitely predispose to infection

The predominant infective organism is one of the *Bacillus coli* group Its occurrence is observed in more than 80 per cent of the cases although staphylococci streptococci and *Bacillus proteus* are occasionally identified The hematogenous mode of invasion is demonstrated by the fact that the bladder changes indicative of infection are comparatively uncommon

Symptoms The symptomatology of pyelonephritis of pregnancy does not differ from pyelonephritis of nonpregnant females The symptoms may vary from that of a very mild form to an acute fulminating form showing severe febrile reactions Frequency of urination may be the only symptom referable to the bladder Usually the triad of symptoms frequency dysuria and urgency will be noted if the bladder is inflamed Examination of the urine reveals the presence of pus cells albumin and bacteria Associated with these vesical symptoms are the more general febrile reactions chills fever sweats nausea and vomiting Pain and tenderness in one or both loins are usually apparent There is no difference in the severity of the symptoms between pyelonephritis of pregnancy and pyelonephritis in nonpregnant women The severity of the infection depends entirely upon the virulence of

the offending organism and the individual reaction against such bacterial invasion.

Diagnosis: Pyelonephritis of pregnancy is more apparent between the ages of 20 and 30 years, the greatest child-bearing period. The methods of diagnosis do not differ from those of other upper urinary tract infections. The history, complete physical examination, routine microscopic urine examination, and urine cultures will give sufficient data for a working yet accurate diagnosis. A complete urological examination should be made if the physical findings suggest an infection.

Cystoscopy: The introduction of the cystoscope during pregnancy may be difficult due to the encroachment of the gravid uterus upon the pelvic viscera (see Fig. 46). Cystoscopy may be the source of considerable discomfort to the patient and it may be unsuccessful at times. The mode of introduction of the cystoscope depends greatly upon the stage of pregnancy. The bladder is caused to assume different positions and contours as pregnancy progresses to term. The introduction of the cystoscope is accomplished in the early months of pregnancy by markedly depressing the ocular end of the instrument, permitting the beak to pass into the cavity of the bladder.

The bladder having been pushed forward and upward, the distance between the internal sphincter and the floor of the bladder is greatly limited. In early pregnancy, the trigone is broadened and elevated by the enlarged cervix, which tends to elevate the floor of the bladder. The ureteral orifices, instead of having their usual position in relation to the interureteral ridge, tend to face laterally on the outer margin of the ureteral ridge. In general, the outline of the trigone is symmetrical, but as pregnancy continues and the bladder is further distorted, the trigone becomes asymmetrical. As pregnancy progresses beyond the fifth month, insertion of the cystoscope becomes more difficult. The bladder is

deformed by pressure exerted by the gravid uterus, the deformity being not only in the anteroposterior direction, but also laterally The trigone is elongated in its anteroposterior plane and is pushed downward so that the distance between the ureteral orifices and the internal sphincter is increased

In the latter months of pregnancy, introduction of the cystoscope becomes more difficult The beak of the instrument must be turned laterally immediately after passing the internal sphincter The instrument is permitted to be advanced into the extended lateral recesses of the bladder by directing the instrument laterally and in a slightly downward direction Only one ureteral orifice may be observed by this manipulation To visualize the other ureteral orifice it is necessary to withdraw the cystoscope into the internal vesical sphincter the same maneuver to be repeated in the opposite direction

Crabtree considers cystoscopic procedure a safe method of examination during any stage of pregnancy and the puerperium He states there is little danger of the interruption of the pregnancy by cystoscopic manipulations He further states "Cystoscopy is sometimes contraindicated in the febrile patients when not pregnant, but is not in pregnant women "

In instances where the symptoms portrayed by the patient point to involvement of both kidneys, bilateral ureteral catheterization is essential If only one side is affected, preliminary ureteral catheterization should be unilateral for fear of introducing infected material into a medium set for infection but as yet sterile Usually the catheter after introduction, will be seen to drain urine freely and continuously after passing the intramural segment of the ureter In other instances drainage through the catheter will not be apparent until after the brim of the bony pelvis is passed In other instances, drainage will not occur until the catheter has entered the renal pelvis

Treatment: The treatment of pyelonephritis of pregnancy differs little from that of the nonpregnant female. The infection can usually be controlled without recourse to radical procedures until the termination of pregnancy, but cannot be cured after delivery. Rest in bed, suitable dietary-regime, normal bowel activity, the administration of alkalies in the event urine cultures show the presence of *Bacillus coli* will make a medium unfavorable for their growth; administration of large quantities of water by mouth, or intravenous administration of saline solution; the maintenance of normal and adequate drainage of the renal pelvis by indwelling ureteral catheter will usually permit progression of pregnancy to a normal delivery.

Urinary antiseptics may be of considerable benefit but should be administered with caution. By far the greatest single factor in the treatment of pyelonephritis of pregnancy is adequate drainage. Such drainage is best maintained by an indwelling ureteral catheter. Pelvic lavage through the indwelling catheter may be advisable. Such solutions as silver nitrate, 1:1000 to 1:5000; acriflavine, 1:2000 to 1:4000, or mercurochrome, one per cent, are suitable for this purpose.

Nephrosis of Pregnancy: Nephrosis of pregnancy, so called, is a degenerative lesion of the renal tubules. Because of associated hypertension, it cannot be classified as a nephrosis, yet the pathological features are such that it cannot be classified as a glomerulonephritis (FIG. 430). Boyd states that . . . "The lesions are degenerative and among the clinical symptoms are edema and massive albuminuria, so that the general picture is nephrotic in type. Since, however, one of the principal features is marked hypertension, it is evident that the condition is not a nephrosis in the ordinary clinical sense of that term." He further believes that most cases, when followed long enough, turn out to be chronic glomerulonephritis. The condition is usually observed during the

last four months of pregnancy. The symptoms presented usually are marked hypertension, edema, massive albuminuria, oliguria and the presence of casts in the urine. There is usually no great retention of blood urea nitrogen. In the more severe cases, convulsions occur and the condition is then known as eclampsia. These eclamptic convulsions are undoubtedly due to some intra

Fig. 430—Kidney—Acute nephrosis (eclampsia). Advanced fatty granular and hyalin degeneration of the tubular epithelium. Note cellular debris in lumen which frequently results in obstruction and the ischemic glomeruli. (Courtesy of Dr. Lawrence W. Smith and Dr. Edwin S. Gault.)

cranial circulatory changes accompanied by hypertension. Arnold believes that fluid imbalance is a most important factor in convulsions of eclampsia. At autopsy, he has noted cerebral edema and congestion as well as intracranial effusions. He has been very successful in the management of eclampsia by the adoption of a definite establishment of the fluid balance and a dehydration principle now well recognized.

The underlying factors producing all the changes observed in eclampsia are not understood. Although there are many theories, none can fully explain its occurrence. Fishberg is of the opinion that eclampsia is the precipitating factor in establishing essential hypertension in a woman constitutionally predisposed to hypertensivevascular disease. Clinically, however, Arnold has observed many women in eclamptic convulsive seizures who have not shown an elevation of blood pressure. He likewise has observed women with such an elevation of blood pressure that the standard sphygmomanometer did not have sufficient range to record the extreme hypertension, yet these women did not have convulsions.

The kidney lesion in eclampsia shows marked tubular degeneration and swelling of the epithelium of the convoluted tubules. Bell believes that the essential renal lesion is a marked narrowing of all the glomerular capillaries due to thickening of the capillary basement membrane. He has also observed many hyalinized glomeruli with varying degrees of tubular atrophy. Weiss observed, in a case coming to autopsy, that there were . . . "pronounced chronic glomerular changes with tubular atrophy. There was some fibrosis of the interstitium replacing the destroyed parenchyma, but there was very little evidence of arteriosclerosis of renal vessels."

Arnold considers three essential and important steps in dehydration for the control of convulsions of eclampsia: (*a*) Spinal drainage, 40 to 80 cc. withdrawal of spinal fluid every three to six hours; (*b*) hypertonic solutions by vein, 50 cc. of 50 per cent glucose or 20 cc. of ten per cent magnesium sulfate solution at three- to six-hour intervals; (*c*) saturated solutions of magnesium sulfate by mouth, stomach tube or rectum. Fluids are withheld from 24 to 48 hours and their administration resumed according to the output and mental status. Feeding is gradually resumed at three-hour intervals and a strict check of the fluid balance is maintained.

Symmetrical Cortical Necrosis of the Kidneys· Symmetrical necrosis of the renal cortex is a rare lesion complicating pregnancy There is an almost complete necrosis of the cortex of both kidneys The condition is characterized by asymptomatic suppression of urine The only portion of the cortex to escape necrosis is a thin layer lying immediately beneath the capsule This portion of the cortex is supplied by anastomosis from the capsular arteries Infection caused by thrombosis of the interlobular arteries is considered to be the cause

The clinical picture resembles that of obstructive anuria except for the complete absence of pain The anuria is usually complete Death quickly ensues

XXX

UROLOGY IN CHILDREN

The urological problems presented by children are more frequently associated with, or caused by anomalies of the urinary tract than those problems observed in adults. The urinary infection that follows stasis, which is secondary to anomalous formation, usually occurs at an early age. The method of the production of urinary stasis and infection may differ slightly from that occurring in adults, but the end results are similar. The instruments employed in the examination of infants and children are, of necessity, small in caliber. Due to the relative size of the patient and the small size of the instruments necessarily employed during the examination or treatment, considerable difficulty may be experienced until the use of the various instruments is mastered.

The successive steps of history taking and physical examination of infants and children differ little from those used in adults.

1. History: The clinical history should be carefully taken. In most instances, the history must be given by the parent or one who is in charge of the child. It should include not only a general resume of all illnesses and idiosyncrasies, but also a minute, detailed description of the individual urological problem or complaint.

2. Physical Examination: The general physical examination should be complete in every detail. Careful attention should be paid to possible foci of tuberculous and nontuberculous infection in the lymph nodes, bones, and joints. Careful, thorough, but

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gentle bimanual palpation of the abdomen for enlargement in the renal area should always be done Any palpable abdominal mass observed in a patient displaying symptoms referable to the genitourinary tract should be carefully studied to affirm or deny the presence of an ectopic position of a kidney of congenital origin

3 Examination of Genitalia: The local examination of the external genitalia must include

(a) Examination of the penis and urethral meatus for various anomalies
(b) Examination of the urethra, male or female
(c) In instances of incontinence, careful search should be made of the vestibule of the urethra and vagina for an ectopically placed ureteral orifice in the female

4 Rectal Examination· Rectal examination in the male should always be included in instances presenting a history of bladder distention or in instances of frequent or difficult urination

5 Urine Examination· Examination of the urine does not differ greatly from the examination of the urine in adults The only difference is the collection of the specimen In older boys a normally voided specimen may be used In girls and in infants of either sex it is advisable to catheterize If a culture is desired, catheterization is necessary in every instance

6 Intravenous Urography· Intravenous urography, as a preliminary diagnostic procedure, may yield important information The functional ability of the kidneys, their position, contour, their relation to abdominal tumors are pertinent data which may be gained by this procedure It may be difficult, in some instances, to inject the contrast material due to the small veins and the lack of cooperation of the patient Satisfactory results have been attained by subcutaneous injection of the contrast medium A method suggested and used successfully by Mullen has been the sub

cutaneous injection of 20 cc. of 35 per cent diodrast in 80 cc. of normal saline solution. The injection is made at the angle of each scapula, using two clysis needles. Each area should be prepared by iodine and alcohol. A small wheal is first raised by a one per cent solution of novocain at the site to be injected. Films are exposed at five-minute intervals following the diodrast injection until three films have been exposed. Subsequent films are then exposed at ten-minute intervals. A total of 50 minutes following injection is routinely used to permit good visualization. Compression of the ureters is not done.

7. Cystoscopic Examination: Complete urologic examination, including cystoscopy, should always be done in the event of continued pyuria or pyuria that recurs in spite of the usual therapeutic means. Cystoscopic examination may be made under local anesthesia in a great many instances. In infants or very young children, general anesthesia is advisable. Much depends on the type of anesthetic and the selection of the proper sized instrument to be used in the examination. There are many types of cystoscopes used in the examination of infants and children. It is necessary that the cystoscope used be of small caliber. The cystoscopist should acquire the skill necessary for the successful use of these instruments with the least amount of trauma. Three cystoscopes of various sizes may be employed for examination and treatment of infants and children. A Butterfield (11 F) single catheterizing cystoscope may be used for infants and young children. A Laidley (16 F) double catheterizing or a Brown-Buerger (16 F) double catheterizing is satisfactory for other children. Both the Laidley and Brown-Buerger instruments have large visual fields and excellent irrigating facilities. The Butterfield instrument may present considerable difficulty because of its optical system until its use is mastered. The visual field of this instrument appears very close

and somewhat magnified The ureteral orifices appear as relatively large openings or slits and they appear to be at or just within the vesical orifice

Cystoscopy in children does not differ from cystoscopy in the adult Children should be placed in a horizontal position for the cystoscopic procedure The buttocks should be at the edge of the cystoscopic table The thighs and legs should be supported by trough supports that permit abduction of the thighs The trough supports are cupped to accommodate the contour of the thigh and leg and are padded for comfort The thighs and legs are held in position within the supports by adjustable web straps

Infants and children tolerate cystoscopy and cystoscopic procedures as well as if not better than adults However their tissues are thin and delicate and are easily traumatized and for that reason if for no other gentleness of manipulation must always be exercised

General anesthesia is advisable for cystoscopy in children under five years of age as well as many older children particularly if they are unruly or of an apprehensive type Venal ether is one of the recent inhalation anesthetics It is easily and readily administered the patient reacts quickly Venal ether may be given at any age Nitrous oxide and oxygen is preferable in the anesthesia of older children Wherever possible local analgesia is preferred Diothane (one per cent) has proven satisfactory The urethral instillation is made slowly and gently great care being taken never suddenly or painfully to overstretch the urethral walls After an interval of five minutes a small soft rubber catheter is introduced into the bladder This is then emptied of its contents and is irrigated until the returned fluid is clear Before the catheter is removed a small quantity of diothane is introduced into the bladder and deep urethra

In the male infant it may be necessary to dilate the urethra be

fore introduction of the cystoscope. Dilatation is best accomplished by woven bougies rather than by metal sounds. Urethral meatotomy may be necessary before dilatation may be done. Gentleness of procedure in dilating or introducing any urethral instrument or cystoscope, even though the infant or child is under anesthesia, should always be exercised.

The introduction of the miniature instruments in infants or children is similar to the introduction of the larger instruments used in adults. As has been stated, the instrument should be guided, not forced, through the urethral canal. Naturally, the distance traversed by the instrument, when being introduced in infants and children, is less than the distance traversed by a cystoscope introduced in an adult. It is essential, in introducing the instrument, not to begin the downward swing of the ocular end of the cystoscope until the triangular ligament is reached. If the forward motion of the cystoscope is momentarily stopped, continuous gentle pressure will relieve the spasm of the external urethral sphincter. The beak of the cystoscope may then be readily advanced into the bladder if normal conditions exist in the posterior urethra. The introduction of the cystoscope may be rendered difficult in the presence of congenital valves of the posterior urethra or contracture of the vesical neck. Do not force the instrument. The presence of pathological conditions may necessitate urethral dilatation before cystoscopy may be accomplished or may even prevent cystoscopy.

Following the introduction of the cystoscope, the urine may be collected for routine analysis or culture. The bladder may be irrigated through the sheath of the cystoscope until the returned irrigating fluid is clear. The cystoscopic telescope is then introduced into the sheath and locked into position. The bladder is distended using a small, graduated, low hung reservoir. The reservoir should not be more than 24 inches above the patient.

The bladder should never be overdistended A syringe should never be used to fill the bladder The quantity of fluid necessary for suitable distention of the bladder for cystoscopy varies according to the age of the patient and the distendability of the bladder In infants, 25 cc is usually sufficient At two years, 25 to 50 cc No more than 75 to 100 cc of fluid should be used for a child eight to ten years old

Ureteral Catheterization In the use of a single catheterizing cystoscope in the male, a ureteral specimen should be first collected from the side considered normal Following collection of the specimen another catheter is introduced into the ureter of the affected side The cystoscope may then be removed The studies desired may then be completed In females, it is possible to introduce a catheter into one ureter, withdraw the cystoscope to free it from the catheter, reinsert the instrument and catheterize the opposite side In older children, whose urethra is of sufficient caliber to permit the use of a double catheterizing (16 F) instrument, the manner of ureteral catheterization is managed as it is with the adult Following catheterization of the ureters, individual specimens may be taken for analysis or culture, or differential kidney function tests may be done Ureteral catheterization should always be practiced, not only as a method of securing individual renal function or individual ureteral specimens but also for the purpose of retrograde pyelography Radiopaque ureteral catheters should also be used as considerable important information may be gained as to the relative position and course of the ureter or the position of the kidney otherwise considered normal

Pyelography Great care should be exercised to avoid overdistention of the infant renal pelves or calices by the opaque medium The most satisfactory pyelographic medium is a 20 per cent solution of skiodan or similarly allied chemical solution The opaque

medium should never be injected through the catheter by syringe. The renal pelves and calices should always be filled with the opaque medium by gravity flow (Fig. 431). The amount of fluid necessary to fill the renal pelves and calices varies with the age of

Fig. 431—Bilateral retrograde pyelogram showing bilateral hydroureter and hydronephrosis.

(Courtesy of Dr. Elmer Hess.)

the patient and the underlying pathological condition that is present. Normally, in infants not more than two cc. should be used; in five-year-old children, not more than four cc.; in eight to ten years, five cc. of the pyelographic solution are sufficient (Fig.

432) A safe axiom to follow in doing retrograde pyelography is—*Never inject by syringe—never overdistend the pelves and calices—always use gravity flow*

Cystography The need or desire to outline the bladder cavity

Fig 432—Retrograde pyelogram Normal renal pelvis in an eight year old child (*Temple University Hospital Acc No 33088*)

by radiopaque solution in children is common It is a necessary procedure in lower urinary tract obstruction The only contra indication to cystography is when the disease of the bladder is acute Cystography is definitely indicated in all instances of chronic urinary tract infection or disturbances of urination in infants and children Cystography should never be done while

the patient is under anesthesia. The bladder should never be over-distended. In infants up to one year of age, not more than 25 cc. of the opaque medium should be used to fill the bladder. In children up to seven years of age, 75 to 100 cc. may be used. At two years of age, 25 to 50 cc. are usually sufficient for proper distention. Children up to eight or ten years will tolerate up to 100 to 120 cc. of the distending opaque medium. The radiopaque cystographic medium preferred is a ten per cent solution of skiodan or allied chemical solutions. A three per cent solution of sodium iodide may be used. The latter solution may be quite irritating to the delicate tissue. The bladder should be thoroughly irrigated following the use of such irritating solutions before the urethral catheter is withdrawn.

CONGENITAL VALVES OF THE POSTERIOR URETHRA

One of the most important types of congenital obstruction in the lower urinary tract in males is that of congenital valves of the posterior urethra. When these valves are present, they are found only in the posterior urethra, usually in close proximity to the verumontanum. These valves are folds of the mucous membrane of the urethra. The folds of mucous membrane are attached in such a fashion as to impede the outward urinary flow. The pathological importance of these congenital valves is demonstrated by the destruction created through the distention of the bladder, ureters, and renal pelves. Campbell states . . . "Except for stenosis of the meatus, valves of the posterior urethra are the commonest congenital infravesical obstruction and are confined to the prostatic canal." This congenital anomaly was originally described by Langenbeck in 1802. The importance of the condition was not recognized until 1919, when Young, Frontz, and Baldwin stressed the pathology of the anomaly when they presented 23 cases

collected from the literature Young classified this anomaly into three types Each type depended upon the situation of the valve in relation to the verumontanum (FIG 433)

Type 1 This is the most commonly observed type It is characterized by a ridge like fold of tissue lying on the floor of the urethra The ridge like folds of mucous membrane produce a dis

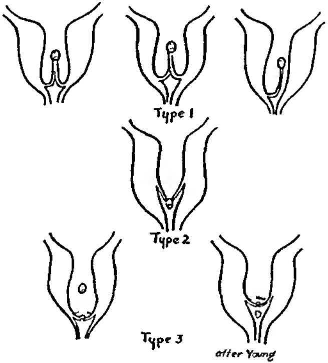

FIG 433—Various types of congenital valves of the posterior urethra

tinct cusp formation This cusp formation is produced by the attachment of the mucous membrane to the anterior end of the verumontanum which also extends to the lateral walls of the urethra This ridge of mucous membrane may produce a single cusp formation attached to one wall of the urethra, or may form a double cusp

Type 2 The ridge or fold of mucous membrane is attached to

the posterior lip of the verumontanum and extends upward through the urethra toward the bladder. The fold is attached to the urethral wall at a point just outside of the internal vesical sphincter.

Type 3. This type is characterized by diaphragms of mucous membrane. These mucous membrane diaphragms may be partially or completely attached to the circumference of the urethra with a minute opening in the center of the membrane. This type of valve may be totally independent in position to the verumontanum and may be found proximal or distal to it.

The etiology of congenital urethral valves is not known. There are several theories but none can fully explain these anomalous formations. Young favors the view of Tolmatschew, who considered them as an enlargement or hypertrophy of normal urethral folds.

Regardless of the type, the distinctive processes produced in the upper urinary tract are similar to those produced by all infravesical obstructions (FIG. 434). The urethra is dilated above the valves. There is also present a dilatation of the vesical sphincter. As the result of back pressure, the bladder wall becomes hypertrophied, later becoming dilated. The continued back pressure causes ureteral and pelvic dilatation with the ultimate formation of hydroureter and hydronephrosis.

Symptoms and Diagnosis: The symptoms are those of infravesical obstruction; that is, dysuria, retention and dribbling of urine. Added to these symptoms is a distended abdomen as the bladder, ureters, and kidneys become dilated. If the obstruction is not eliminated, signs of renal failure appear. Gradually, these symptoms increase in intensity and severity until death ensues. In types 1 and 2, catheterization or instrumentation may be readily accomplished. In type 3, or the diaphragm type, the urethra may be obstructed to all instruments except a filiform. It is pos-

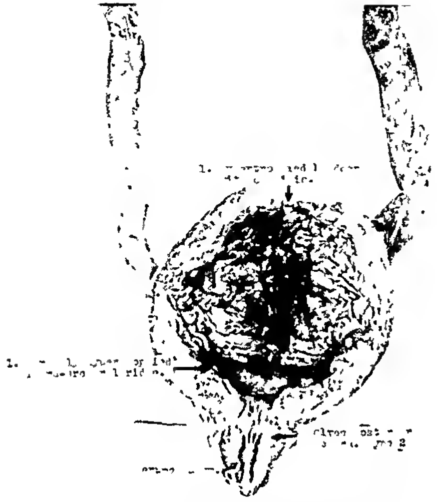

Fig 434—Autopsy specimen. Posterior urethra opened showing type 2 valves Note the hypertrophied bladder, hypertrophied intraureteral ridge and the hydroureters.
(Courtesy of Dr Elmer Hess)

sible, with the type 3 formation, that following dilatation by any instrument, the obstruction is completely eliminated and no further treatment is indicated

The diagnosis is made by intravenous urography and cystography and is confirmed by cystourethroscopy. Cystourethroscopy

will reveal the obstructive folds in the prostatic urethra. Intravenous urography may reveal the presence of poorly functioning kidneys with dilated pelves. Cystography will reveal a greatly dilated, irregular bladder and frequently a complete outline of the greatly dilated upper urinary tract. Campbell calls attention to the characteristic appearance of the vesical outlet. He described this appearance as beginning at the vesical neck and by a tapering dilatation extending through the posterior urethra to the point of obstruction.

Treatment: Gradual decompression of the dilated urinary tract is imperative. Decompression is necessary before complete examination or treatment can be instituted. Decompression may be readily accomplished in those instances where catheterization is possible. In instances where impassable urethral obstruction exists, the bladder may be drained by suprapubic trocar or cystotomy incision. Drainage, regardless of the method, should be continued until there is stabilization of the urinary function. It should be done in a similar manner as stabilization of urinary function in aged individuals suffering with prostatic obstruction. Following stabilization of the urinary function, the removal of the obstructive folds of the urethra is accomplished by the use of fulguration or the cutting current applied by a suitable electrode introduced through the miniature cystoscope.

CONGENITAL HYPERTROPHY OF THE VERUMONTANUM

Congenital hypertrophy of the verumontanum to such a degree as to cause urinary obstruction is infrequently observed. The etiology of this condition is not known. The symptomatology is identical with that produced by congenital valves of the posterior urethra, or any other infravesical obstruction (FIG. 435).

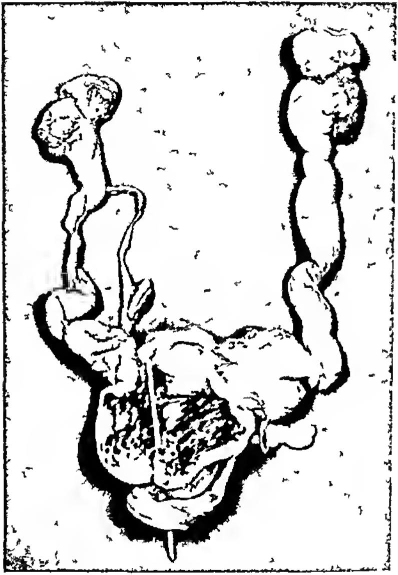

Fig 435—Congenital absence of urethra Note the hypertrophied bladder and hydroureters.
(Courtesy of Dr Elmer Hess)

Diagnosis: The diagnosis is made by cystourethroscopy. The verumontanum is found to be so greatly enlarged as to fill the posterior urethra. In spite of such a degree of hypertrophy of the verumontanum, there is usually little or no resistance offered to the passage of catheter or other urethral instruments.

Treatment: The treatment of congenital hypertrophy of the verumontanum is not unlike that of congenital valves of the posterior urethra. Decompression to permit stabilization of the urinary function is primarily done. Decompression is followed by direct application of the fulgurating current to the hypertrophied verumontanum or the removal of the verumontanum by electro-excision. The removal of three or four full length longitudinal strips of the hypertrophied tissue will usually cause sufficient contraction of the verumontanum to relieve urinary retention and to restore normal function.

CONGENITAL CONTRACTURE OF THE VESICAL NECK

Congenital contracture of the vesical neck is a condition that is also the cause of chronic urinary retention in children. This condition was first described by Beer, who believed that secondary fibrosis of a spastic sphincter occurred in neuromuscular vesical disease. Histologically, a submucous fibrosis of the vesical outlet occurs. The fibrosis may involve the entire circumference of the vesical outlet, or only a portion of it. When only a portion of the sphincteric ring is involved, the lower segment of the ring is the usual site of involvement. In such a location, an atypical median bar formation is produced. The upper or anterior segment of the sphincteric ring is seldom independently involved. Fibrosis of the vesical neck is usually accompanied by hypertrophy of the trigonal musculature. The bladder wall adjacent to the vesical neck is also involved by the extending fibrotic change.

Symptoms: The symptoms produced are similar to those of any infravesical obstruction.

Diagnosis: The diagnosis is made by cystoscopy and cystography. Introduction of the cystoscope may be impossible due to the marked contracture and rigidity of the vesical outlet. If the contracted vesical orifice is of such a size as to permit the introduction of the cystoscope, the instrument may be gripped firmly as it passes through the fibrotic orifice. If the entire vesical sphincter is involved, a rigid circular collar-like intrusion of the orifice will be seen at the time of cystoscopy. If there is only partial involvement, the lower arc or segment of the vesical sphincter will present a picture not unlike that of a median bar formation seen in men. Further cystoscopic observation will reveal the vesical trigone to be prominent and hypertrophied The ureteral orifices will be widely dilated and open. Trabeculations, saccules, and diverticulous formations are not uncommon occurrences. Cystography will reveal a dilated, irregular bladder with or without diverticulous formation.

Treatment: The treatment of congenital contracture of the vesical neck is the enlargement of the vesical orifice. Dilatation may be accomplished with urethral sounds or bougies. In other instances, open cystotomy with resection of a "v-shaped" wedge of tissue from the vesical sphincter may be necessary to relieve the obstructive contraction When removal is necessary, the most ideal methods are the use of the cutting loop technic or the punch procedures as advocated by Young or Caulk.

OBSTRUCTIONS IN THE UPPER URINARY TRACT

The obstructive factors induced by anomalies of the upper urinary tract in children are extensive as well as common in occurrence. Although many children with renal anomalies may advance

to adult life without apparent symptoms, the great majority are subjected in early life to symptom complexes caused directly or indirectly by obstruction occurring secondarily to the anomaly. The pathological changes relative to the urinary tract are limited to the kidney and ureter in combination with the anomalies that exist. The pathological pictures that are presented are in a way similar to those seen when obstruction occurs in the lower urinary tract. The causative factors and the pathology of the lower urinary tract are lacking. When obstructive uropathy is apparent in the lower urinary tract, the urethra, bladder, ureters, and renal pelves are involved, while in obstructive uropathy of the upper urinary tract, the ureters and kidneys only are involved.

The most constant clinical factor in the presence of anomalies is one of infection manifest by long-continued pyuria. Such pyuria is constant and persistent and is not eliminated by the usual therapeutic measures. The frequency with which gross anomalies of the upper urinary tract exist in association with the more trivial genital anomalies should be constantly borne in mind. The presence of pyuria in the presence of genital anomalies (stenosis of the urethral meatus, hypospadias or undescended testicles) should lead one to complete urological examination regardless of the age of the patient.

The anomalies producing such obstruction to the upper urinary tract have been discussed under the anomalies of the kidney and ureter.

URINARY INFECTION IN CHILDREN

As a general rule, renal infection in children does not differ from a similar condition occurring in the adult. The incidence of occurrence of obstructive uropathy is greater in children than in adults. This is undoubtedly due to the fact that stasis secondary to obstructive anomalies produces symptoms early in life. It is also

an established fact that the highest incidence of urinary tract infection seen in infants and children is observed in children under three years of age.

Symptoms: The symptoms of a sudden high temperature, rapid pulse, apathy, restlessness, and gastrointestinal disturbances without localizing renal symptoms, are commonly observed. Due to the general symptomatology without localization and the difficulty of collection of a urine specimen for study, a diagnosis of renal infection has often been overlooked.

A sudden sharp rise in temperature, in the absence of localizing abdominal symptoms, should be considered as of renal origin until proven otherwise. Urinalysis should be done in every instance of unexplained rise in temperature. Further studies may be necessary but much information may be gained by the microscopic study of the urine in the presence of acute symptoms. In the presence of chronic urinary infection, when a history of frequent acute exacerbations is ascertained, congenital urinary obstruction of some type should always be considered. In the more chronic conditions, symptoms referable to the bladder such as frequency and urgency, smarting or burning on urination, may be the only symptoms of which the patient complains. Such history and symptoms warrant complete urological examination.

Diagnosis: The diagnosis of renal infection in children, acute or chronic, does not differ materially from the methods advocated in adults. Urinalysis should be done routinely in the presence of a sudden rise of the temperature from any cause, whether or not there are any localizing renal symptoms. This routine study accounts for the relative accuracy of diagnosis of renal obstruction and infection in hospital practice. Although pus cells may be few in number, their relative infrequency of occurrence should not eliminate the taking of a urine specimen for culture. Many physicians are opposed to urethral catheterization. It is a well estab

lished fact that catheterization, male or female, done under rigid aseptic technic, is justifiable and is not a dangerous procedure. A urine culture should always be done. The causative organism is most frequently the *Bacillus coli,* although staphylococcus or streptococcus or other pathogenic organisms may be found.

Once the presence of a chronic infection of the upper urinary tract is established, cystoscopy and ureteral catheterization should be done. Retrograde pyelographic studies should be done on the affected side. It is routine practice to do intravenous urography before cystoscopy. Considerable pertinent data may be gained by such preliminary study.

Cystoscopy: There is nothing pathognomonic in the appearance of the bladder in infections of the upper urinary tract. There may be few or no vesical symptoms. Acute upper urinary tract infections are usually associated with inflammatory lesions of the bladder. These lesions may be manifest by a slight blush or hyperemia surrounding one or both ureteral orifices. Under such circumstances, vesical symptoms may be slight or entirely absent. There is generally present a localized cystitis confined to the trigone. The trigone is inflamed and may appear edematous. The ureteral orifices appear inflamed and swollen. The inflammatory reaction may be so intense as to obscure entirely the orifices.

The ureteral orifice on the affected side may be surrounded by a congested areola of varying intensity. The vesical mucosa may reveal evidence of a subacute or chronic inflammation.

Indigo-carmine elimination is delayed or absent on the affected side or it may be eliminated in faint concentration. Differential function tests may reveal a drop in percentage elimination on the affected side. Individual ureteral specimens will reveal varying degrees of turbidity of the urine on the affected side.

Treatment: The treatment of renal infections in children is similar to that employed in adults. Acute urinary infections are

usually self-limited and progress to spontaneous cure unless stasis is produced by an anomalous condition.

Treatment includes rest in bed, forcing fluids, active but not violent catharsis and diuresis. In the event of vomiting, five or ten per cent glucose in saline solution should be intravenously administered. Alkalinization of the urine with sodium bicarbonate or potassium citrate, is often efficacious 2.6 to 4 Gm. (40 to 60 grains) per day in divided doses. The use of mandelic acid is somewhat limited, although excellent results have followed its use. Helmholz recommends giving 0.06 Gm. (one grain) of calcium mandelate per each 100 cc. of urinary output in 24 hours. By this means, sufficient concentration of the drug is established as well as the maintenance of a *p*H of the urine of less than 5.5 is assured. Sulfonamide therapy, although widely advocated and extensively used, is not without danger. Under favorable circumstances, sulfadiazine may be used very effectively. It is recommended that one grain per pound of weight be administered. The initial dose should be one-fourth of the total and the remainder divided so as to be given in equal amounts at four-hour intervals during both day and night. It must be borne in mind that a damaged kidney has the power to excrete sulfonamide compounds while impaired kidneys excrete the drug with about the same power as they excrete urea. It is imperative that the blood concentration of the drug be estimated at regular intervals. These intervals between estimation should be no greater than every four days. The greatest factor of treatment is the maintenance of free urinary drainage. Whether in the upper or lower urinary tract, free drainage is essential. Urinary retention, regardless of location, favors infection and must be eliminated.

Treatment of chronic infections: An accurate diagnosis is imperative before intelligent treatment can be instituted. The most frequent causative factor of chronic urinary infection is obstruc-

tion. In chronic infections, examination should include a search for stasis of the upper and lower urinary tracts by cystoscopic and roentgenological examination. The recognition of such stasis should be followed by correction of the obstructive factors. A search for and the elimination of any foci of infection in the body should be made. Constipation should be eliminated. An accurate diagnosis should be followed by a systematic and comprehensive therapeutic regime similar to that used in adults.

UROLITHIASIS IN CHILDREN

Urolithiasis in infants and children, although not rare, is not of common occurrence in America. In certain sections of the Orient particularly India and China, the occurrence of urolithiasis in children is of relative frequency. Thomas and Tanner, in an analysis of 203 instances of calculous disease in children, found that the average age was seven and eight-tenths years. In their series the youngest child was ten months old. Campbell, in a survey of 74 instances (43 post-mortem, 31 clinical) of calculous disease in infants and children, found 36 instances under one year of age; 15 instances at two years of age; eight instances under five years of age, or 59 instances of calculous disease under 15 years of age.

The location of the calculi in the series studied by Thomas and Tanner was 21 per cent in the kidney, eight per cent in the ureters, and 69 per cent in the bladder and urethra. In the series of 35 children coming to autopsy, Campbell found calculi in 29 instances in the renal pelves or calices and two instances at the ureteropelvic junction. No mention is made of the position of the calculi in four instances.

Analysis of the symptoms portrayed in the order of their frequency are pain and colic, hematuria and frequency, pyuria.

dysuria, nausea, and vomiting Many calculi may remain symptomless for years Not infrequently, the most prominent symptom is one of urinary frequency or incontinence of urine It is only by methodical examination that symptomless calculi are discovered

The formation, composition, localization and sequel of urolithiasis in children do not differ from those described in detail elsewhere The treatment of urolithiasis does not differ from that of adults as regards indications and contraindications The greatest difference is that of the size of the patient The surgical procedures are the same In the presence of vesical calculi, suprapubic cystotomy is to be preferred to litholapaxy The latter procedure has been advised by some and miniature lithotrites have been designed Due to the size of the instrument, repeated efforts to crush the stone must be made As in adults, litholapaxy in children is contraindicated in the presence of trabeculations or diverticulum of the bladder

Due to the fact that anomalous conditions of the kidneys favor development of calculi, considerable attention should always be directed to the functional ability, position and contour of both kidneys before surgical treatment to either kidney is instituted

RENAL TUBERCULOSIS IN CHILDREN

Renal tuberculosis in children, although infrequent in its occurrence, is not uncommon Renal tuberculosis is one of those conditions that should be borne in mind in every instance of chronic pyuria that continues in spite of adequate treatment

Tuberculosis in children occurs in the same clinical forms as does the disease in adults the acute miliary and the chronic forms

Acute Miliary Renal Tuberculosis is but a part of a generalized hematogenous tuberculous infection The condition is bilateral does not respond to any known treatment and is rapidly fatal

Acute miliary tuberculosis is commoner in children than in adults. As in adults, the condition is one of medical treatment rather than surgical. The kidney of acute miliary tuberculosis is usually a post-mortem finding because of the rapidity with which these little patients succumb to the overwhelming generalized tuberculous infection.

The kidney cortex is studded with miliary tubercles which present a similar picture to the abscesses of a pyemic kidney because of the multiplicity of the small localized abscesses. There is no border or rim of congestion surrounding the tubercles. The capsule strips readily leaving an intensely congested surface studded with tubercles. On section, tubercles are seen mainly in the cortex but no portion of the kidney is spared. There are no cavities nor any apparent inclination to cavitation as the disease is so rapidly fatal that such pathological changes cannot take place.

Chronic Renal Tuberculosis: The incidence of occurrence of renal tuberculosis is of such frequency that it should always be considered in every instance of chronic pyuria. Tuberculosis in childhood presents the same clinical picture as does tuberculosis in adults. Although tuberculosis in childhood is not as frequent an occurrence as it is in adults, the symptoms of which these little patients complain; the pathological lesions established; the management and surgical treatment do not differ from those in adults. One very definite axiom to follow is that *in the presence of sterile pyuria, tuberculosis should be suspected until proven otherwise.*

Symptoms: Frequent, painful urination is the commonest symptom of which children affected with renal tuberculosis complain. Beginning as frequency of urination during the day and later continued at night, the frequency of urination may be so intense as to cause these patients to void every 15 or 20 minutes. Pyuria is constant. The urine voided is cloudy. It is usually acid in reaction.

It is sterile to ordinary culture Occasionally abrupt painless hematuria occurs Hematuria is never profuse and is usually of short duration Microscopic hematuria may exist for a considerable period before ever being noticed and may be an accidental finding

Associated with these classical symptoms gastrointestinal upsets are frequent These gastrointestinal disturbances secondarily cause loss of weight and strength and anemia of varying degrees

Diagnosis The initial steps in the diagnosis of renal tuberculosis are those of a properly taken history and a complete physical examination Examination of the urine by routine urinalysis may be of valuable aid in suggesting the presence of tuberculosis The absence of renal tuberculosis should never be determined by a single stained smear of the urine sediment The great variations from day to day of the presence of tubercle bacilli in the urine are such that repeated examinations by smear culture and guinea pig inoculations are necessary before a positive diagnosis may be made or excluded

Once a diagnosis of the presence of renal tuberculosis is made the next step is to ascertain whether or not the process is bilateral or unilateral One of the first measures to ascertain the extent of such an infection is by excretory urography Considerable informative data are to be gained by such a procedure the position contour and functional ability of both kidneys may be ascertained as well as the possible extent of the pathological process should it be present Cystoscopy with bilateral catheterization of the ureters and collection of specimens from both renal pelves for culture and guinea pig inoculation should then be done It is also rational to do an individual kidney function test and retrograde pyelography on the affected side while the ureteral catheter is in position All these diagnostic factors have been discussed at length under

renal tuberculosis. The points of diagnosis and surgical procedures do not differ from those practiced in adults.

Treatment: Once a diagnosis of unilateral renal tuberculosis is made, there is no contraindication to surgical treatment that is in any way different from such surgical procedures in similar conditions in adults.

NEOPLASMS OF THE URINARY TRACT IN CHILDREN

Tumors of the kidney in infants and children occur in a greater relative frequency than do tumors of the kidney in adults.

Benign tumors of the kidney in children are rare. Such tumors are seldom of sufficient size to be palpable or cause symptoms. The adenoma, cystadenoma, and fibroma are the usual tumors observed and they are generally observed at autopsy. The adenoma, as previously described, is found beneath the renal capsule. The tumor is usually small in size and may be single or multiple. Occasionally, cystic degeneration occurs with the formation of a cystadenoma. Fibromata also usually lie in a subcapsular position and on occasion may be of such size as to cause symptoms.

The occurrence of renal tumor in infancy and childhood is manifest in those tumors which are malignant. The embryonal adenomyosarcoma (Wilms' tumor) is the most frequently observed malignant lesion of the kidney in infants and children. Hypernephroma is rarely observed in children although it is one of the most frequently observed types of malignant renal neoplasm in adults. Rarely has adenocarcinoma been observed in children. Campbell, in a survey of the literature, could find the record of but two instances of renal carcinoma in children.

Embryonal adenomyosarcoma is the commonest neoplasm of the urinary tract in the young and comprises 20 per cent of all

tumors in children. Such a percentage is second in frequency of occurrence of all tumors in children. The most frequently observed neoplasm in children is that of the eye and orbit, which have an incidence of occurrence of 52 per cent.

The average age of occurrence of embryonal adenomyosarcoma is three years. According to Campbell, more than three-fourths of all renal embryomata appear before the age of five and two-thirds of all the cases before the third year. Hyman, in an analysis of 136 instances of renal tumor in children, found 106 occurred in the first five years; 57 were in the first two years.

Embryonal adenomyosarcoma is usually unilateral but instances of bilateral involvement are recorded. Hinman collected 1037 cases; only four per cent were bilateral. The characteristics of the tumor, pathology, symptomatology, and diagnosis are discussed at length under renal tumors.

Embryonal adenomyosarcoma grow rapidly, the clinical course being about one year. Metastasis is relatively late. The tumors are usually of large size, and for this reason the palpable tumor in the upper abdomen is usually the first sign. Hematuria, if it occurs at all, is usually a late symptom. The composition of the tumor is one of mixed variety. Ewing describes the tumor as . . . "one of isolated tubules of high epithelium or cubical cells with indistinct lamina surrounded by broad zones of indifferent spindle-cells on which is based the diagnosis of adenosarcoma."

Treatment: The consensus of various investigators regarding the treatment of embryonal adenomyosarcoma is that preoperative irradiation, followed by nephrectomy, offers the greatest hope of cure. Such treatment should be instituted early. Unfortunately, the condition is usually seen late in the progress of the disease and as a whole the results attained by operative measures are far from satisfactory. Few instances of five-year cures have been reported.

Nephrectomy should follow within three to six weeks after irradiation. After an elapse of such time, the tumor resumes its active growth. The recurrent growth is less radiosensitive than the original growth and cannot be indefinitely controlled by irradiation.

TUMORS OF THE BLADDER

Tumors of the bladder are very rare occurrences in children. In 1900, Cancetti collected 42 cases and added one of his own. In 1924, Deming reviewed a total of 64 cases found in the literature and added two of his own (FIGS. 436, 437). In 1937, Rathbun reported a personal observation and found ten additional cases in the literature, bringing the total to 75. Rathbun, in his analysis of the cases, found 38 were sarcomata, 16 myxomata, 4 myxosarcomata, 5 fibromata, 2 each of rhabdomyomata, benign polyp,

Fig. 436—Myxoma of the bladder. The patient was a 20-month-old male. Lobulated tumor mass protruding through the bladder incision.

(Courtesy of Dr. Clyde L. Deming and *Surgery, Gynecology and Obstetrics*, 1924, 39:432.)

and hemangiomata 1 each of neurogenic sarcoma fibromyoma neurofibroma leiomyoma dermoid and papilloma

Those bladder tumors occurring in children that are of a malignant nature are usually highly malignant but strangely do not metastasize until late although direct extension to contiguous structures may occur rapidly and relatively early in the continuously advancing progress of the disease

Fig 437—Myxoma of the bladder High power photomicrograph
(Courtesy of Dr Clyde L. Deming and *Surgery Gynecology and Obstetrics* 19 4 39 43)

Symptoms The symptoms of tumor of the bladder in children are those of urinary disturbances frequency dysuria and urgency of urination Hematuria and pain occur later as the disease progresses

Diagnosis The diagnosis of bladder tumor is made by cystoscopic examination and biopsy The cystoscopic picture presented by tumors of the bladder in children is similar to that of vesical growths observed in adults The cystogram may demonstrate irregularities of the vesical outline or a filling defect at the site of

Fig. 437-A—Fibromyxosarcoma of the bladder. Lowpower photomicrograph showing the fibrous nature of the tumor and the apparent polypoid formation.

(Courtesy of the U. S. Army Medical Museum, Acc. No. 69249.)

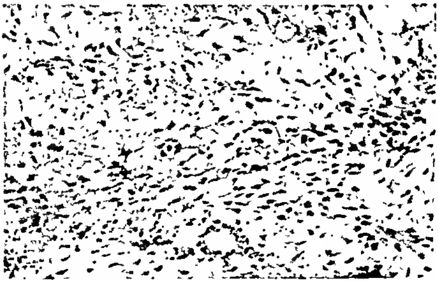

Fig. 437-B—Fibromyxosarcoma of the bladder. Highpower photomicrograph showing the typical stellate cells of the myxoblast and the spindle nuclei of the fibrous series.

(Courtesy of the U. S. Army Medical Museum, Acc. No. 69249.)

the tumor. It remains for the biopsy of the tumor to establish the type of growth that is present (FIGS 437 A, 437-B)

Treatment: Treatment of tumors of the bladder in childhood has been generally unsuccessful. This unsuccessful management is undoubtedly due to the fact that the disease is far advanced when first observed

XXXI

UROLOGICAL ROENTGENOGRAPHY

Roentgenological study of the urinary tract is one of the greatest diagnostic aids at the command of the urologist. The combined use of the cystoscope and the roentgen ray is a perfect combination of diagnostic equipment that permits the most accurate and intricate diagnosis. Previous to the introduction of such equipment, urological diagnosis was mainly one of conjecture. Pyelography or ureteropyelography, either retrograde or excretory, are terms applied to the radiographic demonstration of the renal pelvis or the renal pelvis and ureter respectively (FIG. 438).

Pyelography was first attempted by Klose in 1904. He was unsuccessful due to the viscosity of a bismuth solution he employed. In 1905, Voelcker and von Lichtenberg, while using collargol in doing cystography, accidently demonstrated the outline of the ureter and renal pelvis. The fluid had regurgitated upward through a dilated ureteral orifice. In 1907, Albarran and Ertzbischoff demonstrated the ureter and renal pelvis under normal and pathological conditions. In 1909, Keys advocated the use of 40 to 50 per cent argyrol as a pyelographic medium. Braasch, using a ten per cent solution of collargol, popularized pyelography in the United States. In 1913, Sir Thompson Walker introduced pyelography in England. From that time, urologic roentgenographic studies have become procedures that are universally used. Without the use of such procedures, the high standard of accuracy of diagnosis demanded by modern urological practice could not be maintained.

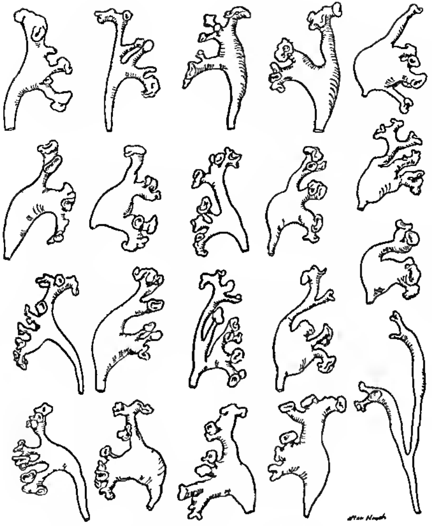

Fig 438—The renal pelvis in the adult.
(Redrawn from Kelly and Burnam After Hauch.)

Excretory or intravenous urography, although an important phase of development, cannot supplant retrograde pyelography (Fig. 439). As an adjunct to cystoscopy and retrograde pyelography or ureteropyelography, the procedure is often invaluable

Fig. 439—Intravenous urogram showing a moderate distention of both pelves due to a bilateral block at the ureteropelvic junction.
(Temple University Hospital, Acc. No 45641)

(Fig. 440). Many factors contribute to the possibilities of faulty interpretation of such roentgenological films. It is for this reason that excretory urography should be considered as an adjunct to urological diagnosis, rather than to rely explicitly on the interpretation of these films.

Urological roentgenologic study should include: (1) The plain

roentgenogram, (2) intravenous or excretory urography, (3) the roentgenogram of the abdomen with opaque catheters within the ureters, but before injection of the opaque medium; (4) retrograde pyelography or ureteropyelography; (5) cystogram.

Fig. 440—Intravenous urogram Normal roentgenogram of both kidneys (Temple University Hospital Acc No 53347)

THE PLAIN ROENTGENOGRAM

The plain roentgenogram or "scout film" of the abdomen may reveal considerable informative data (*Fig 441*). The general bony structure of the lumbar spine and pelvis is clearly visualized Marked deformity or localized areas of rarefaction of the bony

structure are often suggestive of pathological changes which will present urinary symptoms, yet these processes are definitely foreign to the urinary system. On the other hand, areas of increased density of the bony structure suggest definite serious pathological

Fig. 441—Roentgenogram showing a large staghorn calculus that appears similar to the outline of a pyelogram.
(Temple University Hospital, Acc. No. 40692.)

changes in the urinary tract. In those instances of calculous formation, it is usual that urinary calculi are composed of a high percentage of calcium, which is opaque to the roentgen ray and which will cast a definite shadow on the film. The number, relative position, and size of the calculi are usually determined by a plain

roentgenogram The great majority of calculi cast a definite shadow to the roentgen ray Crystallization of uric acid xanthin or cystin with the production of a calculus may cast no shadow or one that is only faintly visible In a perfect roentgenogram it is possible to ascertain the position contour and size of the kidneys as the shadows cast by these organs are of different density than the surrounding structures The outline of the iliopsoas sheath should be clearly visualized A preliminary roentgenogram should precede all retrograde or excretory urographic studies Stereoscopically exposed films should be routinely employed Films exposed by such technic usually afford greater possibilities of positional localization of calculi within the kidney or the relationship of opaque shadows to the kidney

Preparation of the Patient The one important factor which obscures faintly visible shadows is the presence of gas and fecal material within the intestine It is not uncommon in the presence of an acute renal colic that abdominal distention is present The gaseous abdominal distention may be so great as to make difficult the differentiation roentgenographically between renal colic and intestinal obstruction It is usual that some preparation of the patient should be made preliminary to the exposure of the film The use of an enema is routinely employed by many investigators However considerable air may be carried into the lower bowel by such a procedure and although the bowel is emptied of some fecal material it is usual that more air or gas is present following an enema than before it was given

Routinely the use of a thorough purgative is indicated to eliminate the fecal material and gas within the bowel Castor oil has the disadvantage of being poorly tolerated by many individuals The use of saline cathartics has not proven too satisfactory although they are much more pleasant to the taste The use of

saline cathartics is usually followed by an increase of intestinal gas. Castor oil has been found to be the most satisfactory. Two ounces of castor oil should be administered in the afternoon preceding the day of examination. The evening meal should be withheld. All fluids, excepting milk should be permitted until midnight. The roentgen examination is done as early as possible the following morning. Such a routine of catharsis and elimination may be followed by plain roentgenograms or retrograde pyelography or excretory urography. It is usually not necessary to institute catharsis before retrograde pyelography. The shadows produced by the concentrated opaque medium are much more dense and permit better visualization on the roentgenographic film than the shadows produced by excretory urography.

RETROGRADE PYELOGRAPHY

Indications: The indications for retrograde pyelography are rather numerous when it is considered that the kidney and ureter are affected so frequently. There is a wide variety of positions, anomalies, infections, and calculous formation that affect the kidney. The majority of such lesions are demonstrable roentgenographically. In general, retrograde pyelography is indicated whenever a preliminary examination reveals the presence of pus or hematuria. It is also indicated when a history of protracted urinary disturbances is elicited such as frequency and urgency of urination, dysuria or continued nocturia. It is also indicated in those instances where physical examination reveals a palpable mass in the loin with or without urinary symptoms.

Retrograde pyelography is not necessarily required when an accurate and obvious diagnosis can be made without its use. The findings on retrograde pyelography may be termed *proof absolute*.

Data Derived from Retrograde Pyelography The important data ascertained by retrograde pyelography are such as could not be so completely supplied by any other means or method of examination

Fig 442—Roentgenogram showing the position assumed by the ureter in crossed renal ectopia.
(Temple University Hospital Acc No 51294)

1 Detection of congenital anomalies in the upper urinary tract which may be accidentally discovered by pyelography (Fig 442) Yet the anomalous condition may be fundamentally responsible for serious pathological changes which may have produced few symptoms

2 The demonstration of the outline of the ureter as to position size and course taken from the kidney to the bladder (Fig 443)

Many factors influence the contour of the ureter; the flexible radiopaque ureteral catheter will clearly outline this course and position. When the ureter is filled with opaque medium, the size and contour are readily visualized.

Fig. 443—Retrograde pyelogram showing a moderate hydronephrosis with torsion of the hydroureter.

(Temple University Hospital, Acc. No 32707)

3. Demonstration of the outline of the renal pelvis as to position and shape (Fig. 444). A true estimation of the position of the kidney may only be made after filling the renal pelvis. Any undue mobility of the kidney, or whether its position is altered by ex-

trinsic pressure, may be observed. Distention of the renal pelvis, as produced by its numerous causes, is readily demonstrable (Fig. 445).

4. The opaque solution demonstrates the destructive inflam-

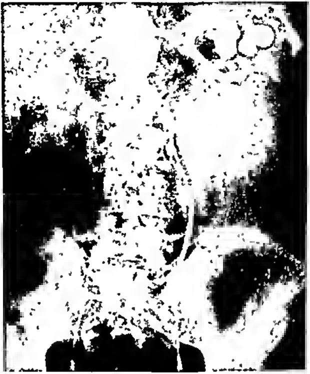

Fig. 444—Retrograde pyelogram. Rupture of pyonephrotic left kidney. Irregular masses of opaque medium may be seen in the upper left quadrant. (Courtesy of Dr. Elmer Hess.)

matory or neoplastic processes of the renal parenchyma, which communicate with the renal pelvis (Figs. 446, 447).

5. The true relationship of the kidney to the surrounding organs is readily demonstrated. Calcified lymph glands or other opaque bodies are readily localized in relation to the kidney. Calcified lymph glands and gallstones are two conditions which are readily confused with renal calculi. It is possible to establish by

retrograde pyelography whether or not the presence of foreign opaque shadows or masses has a definite anatomical relationship to the kidney (Fig. 448).

Contraindications to Pyelography or Ureteropyelography: Retro-

Fig. 445—Retrograde pyelogram—Pyelonephritis. Note unusual contour of renal pelvis.
(Temple University Hospital, Acc No 31143)

grade pyelography is dependent upon cystoscopy. It may be impossible to introduce the cystoscope in the presence of obstructive uropathy of the lower urinary tract. Dilatation or removal of such obstructive factors may be necessary before cystoscopy may be successfully achieved. Retrograde pyelography is not without its dangers and complications. There are definite contraindications

which should not be lightly regarded. Pyelography should never be done in the presence of acute severe illness, general debility or uremia Pyelography is contraindicated in those instances in which blood urea content and renal excretion tests reveal evidences of

Fig 446—Retrograde pyelogram—Carcinoma of kidney Note marked distortion and elongation of lower calyx.
(Temple University Hospital, Acc No. 38021)

renal insufficiency Pyelography should be done very guardedly in the absence of one kidney, either acquired or congenital, or in severe cardiac or vascular disease in which it would be dangerous to do any type of urethral instrumentation.

Contrast or Radiopaque Media: There is rightfully a definite tendency of urologists to discard those toxic or irritating solutions

routinely used in doing retrograde pyelography. Thorium nitrate was discarded because of its toxicity. Sodium bromide was discarded because of its irritating qualities and was replaced by the iodide salts of sodium or potassium. Although universally recog-

Fig. 447—Retrograde pyelogram—Hypernephroma. Note bizarre, crescentic appearance of pelvis and calices.
(Philadelphia General Hospital.)

nized as an efficient agent, potassium iodide is irritating to the tissues. All the toxic or irritating radiopaque media have been replaced by the more recent and less irritating solutions such as skiodan, neo-iopax, diodrast, and hippuran. Although costing more than the older pyelographic media, they are nonirritating, efficient radiopaque media when used in 15 or 20 per cent solutions.

Fig 448—Intravenous urogram showing slight hydronephrosis. Note the irregular narrowing or stricture of upper calyx.
(Temple University Hospital, Acc No. 52679)

Equipment for Retrograde Pyelography: The equipment necessary for retrograde pyelography need not be extensive or elaborate. An adjustable ring stand on a floor pedestal (FIG. 449); a burette clamp; a 50 cc. burette; rubber tubing; a needle adapter; one pinch-cock and a hypodermic needle of such caliber as to fit the catheter are all that is essential. The syringe injection method, using a 20 to 30 cc. syringe, is not recommended.

Anesthesia: The sensation of fullness in the loin, as noticed by the conscious patient, is the best known clinical guide in the filling

of the renal pelvis. General anesthesia is never to be considered whenever pyelography is to be done. The use of intravenous sodium pentothal is ideal for those individuals who demand general anesthesia for cystoscopy. The induction and recovery from the anesthetized state are rapid. All cystoscopic manipulation may be done and the cystoscope removed, permitting the ureteral catheter to remain *in situ* while the patient is in the stage of anesthesia. Following the removal of the instrument, the patient quickly regains consciousness. The injection of the renal pelvis may then be done with the patient fully conscious. Sacral, or low spinal, anesthesia may be used to advantage in those instances that require deeper anesthesia than is afforded by the use of local anesthesia in the urethra. The renal area is not affected by such an

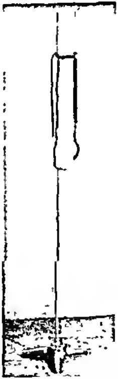

Fig. 449—Standard and burette used routinely in retrograd...

anesthetic The renal pelvis may be filled immediately after introduction of the ureteral catheter if it is so desired

Technic of Retrograde Pyelography. Gravity method of filling the renal pelvis

It is desirable that the entire procedure of cystoscopy and retrograde pyelography be done on a roentgenographic cystoscopic table (Fig 450) If the ureteral catheter cannot be advanced up

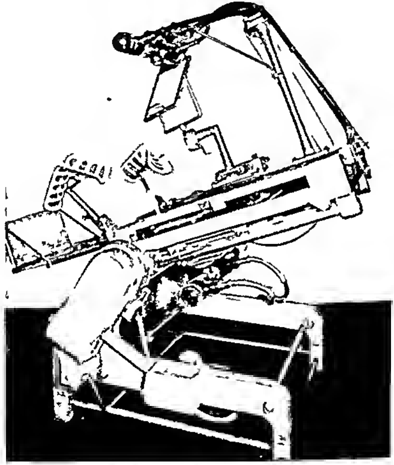

Fig 450—Cystoscopic X ray Table designed by Dr W Edward Chamberlain. This table is equipped for both roentgenogram or fluoroscopy in the horizontal, upright or modified Trendelenburg position.

the ureter more than a few centimeters, the catheter may be pulled from its position during the removal of the cystoscope and the transfer of the patient to an x-ray table. It should be routine practice to expose the x-ray films in stereo after introduction of the ureteral catheters prior to filling the renal pelvis. Gravity filling of the renal pelvis is advocated (FIG. 451). Some prefer injection of the renal pelvis by syringe. It is believed that fewer complications will occur and that pyelography will be a less painful procedure when the filling of the renal pelvis is done by gravity flow.

The head of the cystoscopic table should be lowered so that the

Fig. 451—Retrograde pyelogram. The opaque medium is contained only in the pelvis and calices. No regurgitation along the ureter has occurred. (Temple University Hospital, Acc. No. 42564.)

patient assumes a modified Trendelenburg position All air should be expelled from the rubber tube attached to the burette The rubber connecting tube is attached to the ureteral catheter on the side that pyelography is to be done The burette is adjusted so

Fig 452—Retrograde ureteropyelogram Complete filling of the renal pelvis, calices and ureter is visualized.
(Temple University Hospital Acc No 30961)

that it is no higher than 20 centimeters above the level of the kidney When all is in readiness, the pinch cock is opened and the renal pelvis and ureter are permitted to be filled with the radio paque solution (Fig 452) The patient is cautioned to report any unusual sensation or fullness in the loin Under normal condi

tions, eight to ten cc. of solution will suffice to fill the renal pelvis. Pathological dilatation of the pelvis and ureter may necessitate the use of large quantities of solution to fill completely the dilated channels. Under no circumstances are more than 20 cc. of the

Fig. 453—Retrograde pyelogram. Hydronephrosis of marked extent. Probable cause an aberrant vessel, although it cannot be demonstrated roentgenologically. (Temple University Hospital, Acc. No. 42950.)

radiopaque solution permitted to enter the renal pelvis unless the capacity of the dilated renal pelvis is definitely known.

When the renal pelvis is filled with the required amount of opaque medium, the roentgenographic exposure of the film is made in stereo (FIG. 453). Following the exposure, the head of the

cystoscopic table is elevated so as to bring the patient into a semi erect position. The ureteral catheter is slowly withdrawn while the opaque solution is permitted to run through the catheter under force of gravity (Fig 454). Further films are exposed while

Fig 454—Roentgenogram showing retention within the kidney of the radiopaque medium following the removal of ureteral catheter (compare with Fig 453)
(Temple University Hospital Acc No 4 950)

the patient is in this position. Films taken in the erect position often reveal lesions which might not otherwise be detected, particularly those of movable kidney and kinking of the ureter.

Another method that has proven itself to be very satisfactory in outlining the entire ureter and renal pelvis is the introduction of

a ureteral catheter having a bulbous enlargement on its outer surface, which is of sufficient diameter completely to occlude the orifice. The ureter and renal pelvis may be completely filled without loss of the radiopaque medium from the ureter into the blad-

Fig. 455—Retrograde ureteropyelogram. Stricture of ureter. Dilatation of entire ureter and renal pelvis is evident.
(Temple University Hospital, Acc. No 51659)

der. Another catheter that may be similarly used is a tapered Garceau catheter. This latter catheter has the disadvantage of passing up the ureter for a distance of several centimeters before total occlusion of the orifice is afforded (Fig. 455).

The filling of one renal pelvis at one time is advocated (Fig.

456) If further study of the opposite kidney is necessary, the injection of that organ is done after 48 to 72 hours Many advocate simultaneous injection of both kidneys Others suggest the injection of one side and the taking of the roentgenogram Following

Fig 456—Retrograde pyelogram showing a hydroureter and hydronephrosis. (Philadelphia General Hospital)

the roentgenographic exposure evacuation of the opaque medium through the ureteral catheter is permitted The opposite kidney is then filled with the opaque medium It is believed that the most safe pyelographic technic to follow is the filling of one renal pelvis with subsequent filling of the opposite side in 48 or 72 hours if such study is necessary It is likewise believed that the filling of

the renal pelvis of a solitary kidney (congenital or acquired) should never be done except in very guarded instances. Any roentgenographic evidence should be procured by intravenous urography (FIG. 457).

Fig. 457—Roentgenogram showing a large staghorn calculus completely filling and outlining the renal pelvis and calices. Attempt is being made to do a retrograde pyelogram. None of the radiopaque medium could enter pelvis. (Temple University Hospital, Acc. No. 40692.)

Syringe method: The technic of pyelography by the pressure syringe method is routinely and successfully used by many surgeons. It is not recommended as a routine procedure. The procedure is simply done. A 20 cc. Luer syringe, filled with the opaque medium, is connected to the ureteral catheter. By slow, steady

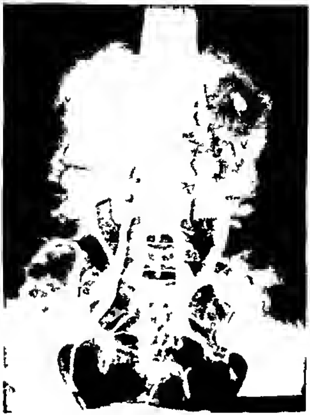

Fig 458—Retrograde pyelogram showing a hydronephrosis and pyelovenous backflow The fan like radiation of the opaque medium may be readily observed. (Temple University Hospital Acc No 27870)

pressure the renal pelvis is filled with the contrast medium and the roentgenographic exposure made The great danger of the method is pain in the renal region The pain is more frequently experienced by the patient than when gravity is employed (Fig 458)

Complications of Pyelography Overdistention of the renal pelvis by the pyelographic media is followed by acute pain in the lumbar region or the side injected Usually this pain is relieved by a hot tub bath or the use of a hot water bottle If the pain is severe morphine sulfate 10 mg ($1/6$ grain) by hypodermic injection

Fig. 459—Retrograde pyelogram. Pyelovenous backflow. (Philadelphia General Hospital.)

may be necessary. A combination of codeine sulfate, 30 mg. (½ grain) and acetosalicylic acid, 0.3 Gm. (5 grains), may be sufficient to relieve the pain.

The use of a large ureteral catheter or one that is eroded or cracked may produce considerable trauma to the ureter (Fig. 459). Complete anuria occasionally develops following simultaneous injection of both renal pelves (Fig. 460). The author has observed such a case: A young woman complaining of pain in the back was subjected to bilateral pyelography. Sodium iodide, 12.5 per cent solution, was used as the contrast medium. Total anuria devel-

oped from which she never recovered Although bilateral pyelography is recommended by many surgeons anuria is a serious complication of pyelography and should be avoided The greatest safeguard against such a complication is unilateral pyelography

Fig 460—Bilateral retrograde pyelogram Tumor of kidney Note distortion of *right renal pelvis caused by a tumor mass in the upper pole of the kidney*
(Temple University Hospital Acc No 3059?)

INTRAVENOUS OR EXCRETORY UROGRAPHY

Excretory urography is accomplished by the intravenous or subcutaneous injection of certain nontoxic compounds which are excreted by the kidneys in sufficient quantities and of sufficient

density to cast an outline of the urinary tract on a roentgenogram (Fig. 461).

There are many factors which contribute to the procurement of a suitable roentgenogram following the intravenous adminis

Fig. 461—Intravenous urogram—Normal. Note normal filling of pelves, calices *and ureters. The excretory function is excellent.*
(Temple University Hospital, Acc. No 62618)

tration of the compound. The roentgenograms obtained by excretory method are not as sharply defined as those obtained by retrograde pyelography. Considerable important information, positive or negative data, may be gained by the routine use of such

procedure as a preliminary study. Excretory urography definitely does not replace cystoscopy and retrograde pyelography, but is of definite material aid in diagnostic accuracy. It is a perfect medium of diagnostic accuracy when symptoms suggest renal pathology or in those instances where cystoscopy and retrograde pyelography are impossible. It is the author's routine practice to do intravenous urography prior to retrograde pyelography in obvious surgical renal disease and routinely before prostatectomy. Visual evidence

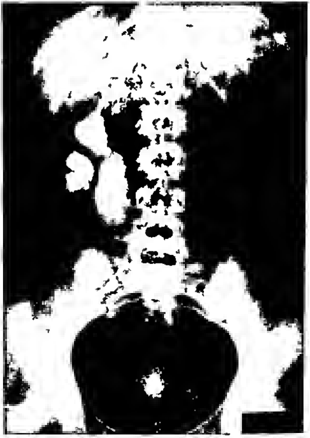

Fig. 462—Hydronephrosis. Intravenous urogram showing hydronephrosis resulting from obstruction at the ureteropelvic junction.

(Temple University Hospital.)

is established as to the position, size, contour, presence or absence of obscure congenital anomalies and the functioning ability of the kidneys. All are salient and important factors in arriving at a true and accurate diagnosis (FIG. 462).

Contraindications: The contraindications to excretory urography are few but very definite:

1. *Intolerance to the drug:* To date, a total of nine deaths have been reported following the intravenous administration of iodine compounds. Granted, this is a small figure if compared with the total number of examinations that have been made, yet it is believed that there would have been fewer deaths if preliminary tolerance tests had been made. The preliminary tolerance tests are easily done. These tests should be done routinely before intravenous injection is made. Two cc. of the iodine pyelographic solution are held in the mouth by the patient for ten minutes. If no reaction occurs, the patient is instructed to swallow the solution. The patient remains under observation for another half-hour. For fear of a delayed reaction to the drug, intravenous urography is not done until after a lapse of 24 hours. Urography is not done if the patient shows any evidence of uticaria, nausea or vomiting, or any other reaction.

2. *Marked renal insufficiency:* It is considered that the kidneys eliminate relatively 90 per cent of the iodine compound. If renal impairment is severe, as revealed by blood urea determination, intravenous urography should not be undertaken.

3. *Impaired liver function:* The liver presumably excretes ten per cent of the drug under normal conditions. If renal function is also impaired, the kidneys are unable to excrete their considered proportional amount and a greater load is thrown upon the impaired liver.

4. *Hyperthyroidism:* Iodine or iodine compounds are never well tolerated in the presence of an overactive thyroid. Opinions

differ regarding the use of the more recent contrast media because of their rapid elimination by the kidneys However, the administration of these compounds should be very guarded in the presence of thyroid disease

5 *Heart disease* Excretory urography is poorly tolerated in the presence of advanced heart disease, an acute febrile condition or severe renal infection

6 *Asthma* Patients suffering with asthma tolerate excretory urography poorly Severe reactions may follow the intravenous administration of the contrast media

Media Used for Excretory Urography Solutions of the nontoxic iodine compounds commonly used at present are diodrast diodrast compound and neo iopax The amount used is relatively the same in each instance All are efficient

Preparation of the Patient Castor oil, two ounces, should be administered in the afternoon (about three o clock) preceding the day of examination The evening meal should be withheld All fluids, excepting milk, are permitted until midnight It is imperative that all fluids be withheld for several hours prior to the roentgenological examination, otherwise the contrast media will be so diluted by the fluids within the urinary tract that sufficient concentration to render a visible shadow will not be gained

Technic of Excretory Urography Two roentgenograms are taken in stereo before the injection of the iodine compound is made A compression bag or pad is strapped over the lower abdomen in such a fashion as to compress the ureters at the brim of the pelvis Twenty cc of the selected contrast medium, to which the patient has previously been tested are injected intravenously The solutions most universally used are diodrast 35 per cent, diodrast compound 40 5 per cent or neo iopax 50 per cent All these compounds have been tested clinically and give relatively similar results

A series of stereoscopic roentgenographic exposures is made. The first films are exposed five minutes after injection of the contrast medium. Under normal conditions, five minutes are sufficient time for excretion of enough contrast medium to outline the renal pelvis. In some instances, the outline of the pelvis will be complete, while in others only a faint shadow will be observed. The roentgenographic films are developed immediately and are used as a guide for further exposures. Usually a second exposure is made 15 minutes after injection of the contrast medium. A third exposure is made 30 minutes after injection. In the event of delayed excretion, exposures may be made in 45 and again in 60 minutes after injection. If desired, the patient may be placed in an erect position, the abdominal compression released and a last exposure made to demonstrate the presence or absence of renal ptosis.

As delineation of the renal pelves, calices, and ureters is dependent upon the excretion of the opaque medium, it may be necessary to make roentgen exposures several hours following injection in those instances where impaired renal function exists.

Urography in Infants and Children: It is frequently difficult to gain parental consent for cystoscopy in infants and children who require urography. Because of the small veins and lack of cooperation of the patient, intravenous urography may be a difficult and tedious procedure. Subcutaneous injection of the contrast medium, under such circumstances, gives excellent results. Mullen suggests the following technic: "The skin is prepared with iodine and alcohol. A small wheal is raised with novocain, one per cent solution, injected into the region of the angle of each scapula. A small nick is then made in each area with scalpel and two clysis needles are inserted subcutaneously. The contrast medium, 80 cc. normal saline, to which 20 cc. of 35 per cent solution of diodrast

have been added, is introduced. The needles are removed and collodion applied. A lollipop is used to keep the child's mouth occupied. Sedatives are not employed A plain or scout film is made before commencing the injection No set rule is followed in the subsequent films Usually, however, films are made at five minute intervals following the injection of the dye until three films are taken and then at ten minute intervals, completing the examination in about 50 minutes following injection. All films are developed as soon as exposed, which furnishes a guide to the intervals between exposure Ureteral compression is not used."

Fig. 463—Cystogram. Diverticulum of bladder Good visualization of the viscus A three per cent solution of sodium iodide was used as the contrast medium.

(Temple University Hospital Acc No. 5[illegible])

Fig. 464—Cystogram. Diverticulum of bladder. Note the relative size of the diverticulum to that of the bladder.
(*Philadelphia General Hospital*)

CYSTOGRAPHY

Cystography is usually dependent upon urethral catheterization. The bladder is filled with an opaque medium and the roentgen exposure made. Following the exposure, the fluid may be voided by the patient and a second roentgen exposure made. This procedure will demonstrate any retention within the vesical cavity of the opaque medium. The shaggy outline of a space-taking lesion is invariably indicative of malignancy. The outpouchings of the vesical wall in the presence of a diverticulum (Fig. 463) are readily noted. The smooth, globular, space-taking outline of a ureterocele may be likewise visualized (Fig. 464).

Technic of Cystography: A urethral catheter is introduced under sterile conditions into the bladder. The injection of the radiopaque solution is made while the patient is on the flat table used in routine roentgenographic work. The bladder is emptied of its contents and a three per cent solution of sodium iodide, or a 20 per cent solution of skiodan is instilled through the catheter. The amount of fluid used is that necessary to fill the vesical cavity without pain. The roentgen exposure is made in the anteroposterior plane and in the oblique positions. Following the roentgen exposures, the radiopaque solution is withdrawn through the catheter and the bladder irrigated with boric acid or normal saline solution.

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CHAPTER XVIII

KIDNEY ANOMALIES

RUPTURE OF THE KIDNEY

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CHAPTER XXIII

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CHAPTER XXIV

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CHAPTER XXVIII

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CHAPTER XXIX

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